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## **Emotional processing and psychosocial factors in individuals diagnosed with dissociative seizures**

Pick, Susannah

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**Emotional processing and psychosocial  
factors in individuals diagnosed with  
dissociative seizures**

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**Institute of Psychiatry, Psychology & Neuroscience  
King's College London**

**2015**

Thesis submitted in partial fulfilment of the requirements for  
the degree of Doctor of Philosophy

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## **Acknowledgements**

I would like to sincerely thank all of the participants who gave up their time to contribute to the research presented in this thesis. I am also very grateful for the funding provided by the Institute of Psychiatry, Psychology and Neuroscience (KCL), and the Central Research Fund (University of London), without which this research would not have been possible.

Importantly, I wish to express my warmest thanks to Professor Laura H Goldstein, for all of the support and input that she has provided as my primary supervisor. Her commitment to the role has been remarkable, and her timely and thorough feedback on my work has been invaluable throughout my time as her student. I consider myself very fortunate to have received her guidance.

I am also very grateful to my second supervisor, Dr John Mellers, for all of his input, particularly the important clinical and theoretical insights that he has provided. In addition, I would like to thank the administrative and medical staff at the Neuropsychiatry outpatients (Maudsley Hospital) and Psychological Medicine (King's College Hospital) departments, for their assistance during recruitment of the clinical sample.

Personally, I am grateful to the friends, colleagues, and family members who have helped me through the many ups-and-downs of juggling this endeavour with work and family-life. I am particularly indebted to those who are closest to me, for providing so much support, encouragement, and commitment during this time. Scott, thank you for everything, particularly your consistent belief that I could do this. To my parents, for all of your support throughout the 'PhD' years and beyond, thank you. Last but not least, an enormous thank you to my daughters, Eve and Ellie, for always understanding when I needed to be at my desk, believing in me wholeheartedly, and never failing to put a smile on face.

### **List of commonly used abbreviations**

DS - dissociative seizures  
ES - epileptic seizures  
AEDs - anti-epileptic drugs  
EEG – electroencephalography  
MRI – magnetic resonance imaging  
HRV – heart rate variability  
ANS – autonomic nervous system  
HPA – hypothalamic-pituitary-adrenal  
MUS – medically unexplained symptoms  
BPD – borderline personality disorder  
PTSD – post-traumatic stress disorder  
ED – emotional dysregulation  
MDI – Multiscale Dissociation Inventory  
IASC – Inventory of Altered Self-Capacities  
FES – Family Environment Scale  
PDS – Post-traumatic Diagnostic Scale  
HADS – Hospital Anxiety and Depression Scale  
TEC – Traumatic Experiences Checklist  
SDQ – Somatoform Dissociation Questionnaire  
IAPS – International Affective Picture System  
ANOVA – Analysis of Variance  
ANCOVA – Analysis of Covariance  
AB – attentional bias  
SCR – skin conductance response

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## **Abstract**

The thesis presents research exploring emotional processing and psychosocial factors in individuals diagnosed with dissociative seizures (DS). Initially, clinical, biological, and psychosocial correlates of DS are discussed, in addition to theoretical perspectives on the disorder. This is followed by a review of previous research investigating dissociation and emotional processing in DS. A novel model of the triggering mechanism underlying DS is proposed, and the rationale, aims, general hypotheses and overall methodology are outlined. Four empirical quantitative studies are then described, in which the DS group were compared to healthy control participants.

A study of psychosocial factors, based on self-report questionnaires, generally confirmed/extended findings from previous studies. Patients with DS reported elevated depression, anxiety, post-traumatic symptoms, borderline personality features, and psychological and somatoform dissociation. Higher rates and greater impact of adverse life events were also reported by the DS group. Contrary to expectations, no group differences in childhood family functioning were observed.

Another study utilising an emotional Stroop paradigm revealed the presence of a preconscious attentional bias towards emotional facial expressions in DS patients. A related experimental investigation of explicit facial affect processing elicited evidence of a deficit in facial expression recognition in the DS sample, alongside reduced autonomic responding to the stimuli in a subgroup of patients. A further experimental study explored patients' affective responses to visual images. Whilst there were no group differences in subjective emotional responses on this task, autonomic responding was elevated in a subgroup of patients.

Qualitative techniques were used in the final study, which explored emotional experiences in a subsample of patients. The findings provided phenomenological insights into patients' understanding of these processes. The findings are drawn together and discussed, and the proposed triggering model is presented in a modified form in the final chapter.

## **Chapter 1. Dissociative seizures: the clinical context**

### **1.1. Introduction**

Dissociative seizures (DS) are discrete episodes of disrupted awareness, sensation, cognition, emotion, and behaviour that resemble epileptic seizures (ES), but are not associated with the organic aetiology of that condition, or any other specific or consistent organic pathology. Instead, DS are proposed to have a psychological causation, generally understood to reflect psychological/emotional distress and to operate at an unconscious/involuntary level (Alsaadi & Marquez, 2005; Bowman, 1998; Fritzsche et al., 2013; Iriarte et al., 2003; Lesser, 2002; Oto & Reuber, 2014; Reuber, 2009). Many patients with DS experience considerable distress and loss of functioning as a result of their attacks (Binder & Salinsky, 2007; Krahn et al., 1997; Lempert & Schmidt, 1990), in addition to undergoing numerous medical investigations/procedures and prescription of anti-epileptic drugs (AEDs).

A variety of terms have been used to describe DS; including 'hysterical seizures', 'non-epileptic seizures', 'pseudoseizures', 'non-epileptic attack disorder', 'psychogenic (non-epileptic) seizures', 'conversion seizures', 'stress-related seizures', and 'dissociative convulsions'. There has been considerable debate regarding the most appropriate label for these phenomena (Benbadis, 2010; Cowan, 2010; Desai, Porter, & Penry, 1982; Gates, 2002; Karam, 2010; LaFrance, 2010; O'Hanlon, Liston, & Delanty, 2012; Sethi, 2010). Some of the terms can be interpreted as pejorative (e.g. 'pseudoseizure'), suggestive of symptom feigning, or can be confusing for patients and carers (Bodde et al., 2009; Scull, 1997; Stone et al., 2003). Currently, the most commonly used label in the literature is 'psychogenic non-epileptic seizures' (Schmutz, 2013). From the patients' perspective, Stone and colleagues (2003) report that general neurology outpatients preferred the terms 'functional seizures' and 'stress-related seizures'.

Within the International Classification of Diseases – 10<sup>th</sup> Edition (ICD-10; World Health Organisation, 2010), DS (termed 'dissociative convulsions') are categorised as a dissociative disorder (along with other conversion disorders), characterised by "a partial or complete loss of the normal integration between memories of the past,

awareness of identity and immediate sensations, and control of bodily movements”. Dissociative disorders are purported to be of psychological origin, and closely related to trauma, insoluble problems and relationship disturbances. DS are included in the category alongside dissociative amnesia, fugue, stupor, trance and possession, dissociative motor disorders, dissociative anaesthesia and sensory loss, multiple personality disorder, Ganser's Syndrome, and unspecified dissociative disorder. These diagnoses require the exclusion of other medical, neurological or psychiatric causes.

However, DS are categorised within the ‘somatic symptom disorders’ group in the Diagnostic and Statistical Manual of Mental Disorders - fifth edition (DSM-5; American Psychiatric Association, 2013), along with other conversion disorders. The somatoform disorders group engulfs a wide range of different symptoms that are indicative of physical/organic illness, but without any clear sign of organic causation (e.g. gastrointestinal problems, fatigue, pain, and genitourinary problems). Somatoform disorders are sometimes referred to as ‘functional’ or ‘medically unexplained’. The commonality between the various somatoform disorders is the presence of medically unexplained physical symptoms that are presumed to be produced by unconscious mechanisms.

More specifically, conversion disorders are those involving symptoms that affect voluntary, motor, or sensory function and mimic neurological conditions, with no organic basis. These include weakness/paralysis, impairment of hearing/sight, aphonia, fixed dystonia, gait problems, and tremor, among others. There has been considerable debate regarding the classification of DS and conversion disorders more generally, and whether or not they should be classified as somatoform or dissociative disorders (R.J. Brown et al., 2007; Guz et al., 2003; Harden, 1997; Kihlstrom, 2005; LaFrance & Zimmerman, 2010; Nijenhuis, 2001; Ozcetin et al., 2009). It has been proposed that a shared psychological mechanism might underlie the conversion (including DS) and dissociative disorders (e.g. R.J. Brown et al., 2007; Kihlstrom, 2005). In the present thesis, this proposed mechanism is referred to as ‘dissociation’, and is characterised by an *involuntary reduction/loss of normal control or awareness of bodily or*

*psychological processes* (see Chapter 3 for further discussion). As such, the term 'dissociative seizures' (DS) is used throughout.

The use of the term 'dissociative seizures' also conforms most closely to ICD-10 terminology, and therefore, that which is internationally recognised. The term DS is used instead of 'dissociative convulsions', to reflect the fact that many patients with the disorder do not have convulsive episodes (see section 1.3). The abbreviation 'DS' is used synonymously with the following: psychogenic/non-epileptic seizures, pseudoseizures, hysterical seizures, dissociative convulsions, functional seizures, conversion seizures, and non-epileptic attacks. The authors of publications cited in the thesis may have used any of the above terms or others; however, the term 'DS' is used for consistency.

The current chapter provides a general overview of clinical issues relating to DS, including symptoms, diagnostic criteria/procedures, epidemiology, treatment approaches and outcomes. The review is based on electronic searches of the following databases: Web of Knowledge/Web of Science, Science Direct, PubMed/Medline and Embase (last updated in January 2015). Three search terms were entered into each database as follows: 'psychogenic seizures', 'dissociative seizures' and 'non-epileptic seizures'. These terms were selected as three of the most commonly used labels for the disorder. Abstracts for original research articles, reviews, and case studies/series were screened for relevance to the current chapter. Articles pertaining to aetiological factors, theories/models and emotional processes in DS are discussed in subsequent chapters. The focus of the review is on patients with adult-onset DS.

## **1.2. Epidemiology and demographic characteristics**

### **1.2.1. Incidence and prevalence**

The incidence of DS has been reported to lie between 0.91-4.9/100,000 per year (Duncan, Razvi, & Mulhern, 2011; O'Sullivan et al., 2007; Sigurdardottir & Olafsson, 1998; Szaflarski et al., 2000). However, most of these studies required evidence of



DS based on video-electroencephalography (video-EEG) monitoring, and so it is possible that these figures may underestimate the actual incidence of DS.

To date, there are no studies detailing the exact prevalence of DS in the general population; however, an estimate based on the available data indicates that it falls between 2 and 33 per 100,000 (Benbadis & Hauser, 2000). However, it is known that DS occurs commonly among individuals assessed in specialist neurological settings. Francis and Baker (1999), for example, summarised a number of epidemiological studies of DS in specialist epilepsy centres and quoted prevalence rates ranging from 9-50%.

### **1.2.2. Gender**

Females are known to be over-represented among adult patients with DS (Rosenbaum, 2000; Schmitz, 2010), with many studies reporting 65-75% of samples being women (Abubakr, Kablinger, & Caldito, 2003; Ahmedani et al., 2013; Alper et al., 1993; Arthuis et al., 2014; Asadi-Pooya, Emami, & Emami, 2013; Buchanan & Snars, 1993; Dixit et al., 2013; Elliott & Charyton, 2014; Ettinger, Devinsky, et al., 1999; Hendrickson et al., 2014; Hovorka et al., 2007; Hubsch, Baumann, & Maillard, 2011; Koby et al., 2010; Krahn et al., 1997; Kuyk, Swinkels, & Spinhoven, 2003; Mitchell, Ali, & Cavanna, 2012; Moore & Baker, 1997; O'Brien et al., 2015; Portuguese et al., 2007; Reilly et al., 1999; Reuber, Howlett, et al., 2007; Scheepers et al., 1994; Selkirk et al., 2008; Silva et al., 2001; Snyder et al., 1994; Thomas et al., 2013; Zhang et al., 2009).

In fact, a considerable proportion of investigators have reported proportions of females that are over 75% (Aboukasm et al., 1998; Alessi & Valente, 2013; Alessi et al., 2013; Bodde et al., 2007; 2013; Bowman, 1993; Bowman & Markand, 1996; Chabolla & Shih, 2006; D'Alessio et al., 2006; Driver-Dunckley et al., 2011; Duncan et al., 2011; Ettinger, Dhoon, et al., 1999; Ettinger, Weisbrot et al., 1999; Gambini et al., 2014; Gates et al., 1985; Gazzola et al., 2012; Kaplan et al., 2013; Karakis et al., 2014; Kristensen & Alving, 1992; Kuyk et al., 2008; Lancman et al., 1993; J.P. Lazarus et al., 2003; Marchetti et al., 2009; Myers et al., 2012; Patidar et al., 2013; Reeves et al., 1998; Reuber, House, et al., 2003; Reuber, Pukrop, Bauer, et al., 2003; Reuber,

Pukrop, Mitchell, et al., 2003; Reuber, Pukrop, et al., 2004; Scévola et al., 2013; Schramke et al., 2010; Uliaszek, Prensky, & Baslet, 2012; van Merode et al., 1997; Wadwekar et al., 2014). Interestingly, a study carried out in China did not find the same predominance of females in their sample, with a ratio of approximately 1:1 male to female (An et al., 2010). This suggests that cultural factors might interact with gender in the aetiology or reporting of the disorder (Schmitz, 2010).

### **1.2.3. Age**

In patients with onset in adulthood, DS typically first occur in the third or fourth decade (Alessi & Valente, 2013; Alessi et al., 2013; Arain et al., 2007; Arthuis et al., 2014; Bowman, 1993; Carton, Thompson, & Duncan, 2003; Dhiman, Sinha, Rawat, Harish, et al., 2013; Dixit et al., 2013; Duncan et al., 2011; Elliott & Charyton, 2014; Ettinger, Devinsky, et al., 1999; Ettinger, Dhoon, et al., 1999; Gambini et al., 2014; Hendrickson et al., 2014; Hubsch et al., 2011; Koby et al., 2010; Krahn et al., 1997; Kuyk et al., 2003; 2008; LaFrance & Syc, 2009; Moore & Baker, 1997; Patidar et al., 2013; Reuber, Pukrop, Mitchell, et al., 2003; Reuber, House, et al., 2003; Reuber et al., 2004; Reuber, Howlett, et al., 2007; Schramke et al., 2010; Silva et al., 2001; Tojek et al., 2000; Wolf et al., 2015).

Age of onset is typically later in patients with DS than those with ES (Berkhoff et al., 1998; M.C. Brown et al., 1991; Derry & McLachlan, 1996; Dixit et al., 2013; Dodrill, 2008; Drane et al., 2006; Elliott & Charyton, 2014; Fargo et al., 2004; Frances, Baker, & Appleton, 1999; Gale & Hill, 2012; Hendrickson et al., 2014; Hixson et al., 2006; Hoepner et al., 2014; Holman et al., 2008; M.D. Holmes, Dodrill, et al., 2001; Karakis et al., 2014; Koby et al., 2010; Litwin & Cardeña, 2000; Locke et al., 2006; Proença et al., 2011; Schramke et al., 2010; Storzbach et al., 2000; Strutt et al., 2011a,b; Szaflarski et al., 2003; Testa et al., 2011; Tojek et al., 2000; Wolf et al., 2015; Zhang et al., 2009).

Whilst diagnoses of DS are less common in childhood and older age, DS do occur throughout the lifespan. A considerable number of studies of DS in paediatric patients have been published (Baker, Moore, & Appleton, 1995; Bhatia & Sapra, 2005; Carmant et al., 1995; Desai & Talwar, 1992; Dhiman, Sinha, Rawat, Vijaysagar, et al., 2013; Gudmundsson et al., 2001; G.L. Holmes et al., 1980; Kim et al., 2012; Kotagal et al.,

2002; Kramer et al., 1995; Kutluay et al., 2010; Lancman et al., 1994; McLean & Dyer, 2003; Metrick et al., 1991; Pakalnis & Paolicchi, 2000; 2003; Pakalnis, Paolicchi, & Gilles, 2000; Patel et al., 2007; Plioplys et al., 2014; Rawat et al., 2014; Salpekar et al., 2010; Say et al., 2014; Selbst & Clancy, 1996; Szabo et al., 2012; Vincentiis et al., 2006; Yi et al., 2014; Yilmaz et al., 2013). The occurrence of DS in older patients has also become increasingly recognised (Abubakr and Wambacq, 2005; Behrouz, Heriaud, & Benbadis, 2004; Duncan et al., 2006; Kellinghaus et al., 2004).

#### **1.2.4. Culture**

Much of the previous literature on DS originated in the western industrialised societies of the United States (US), United Kingdom (UK), and western/northern Europe. However, DS is not specific to this cultural context. There have been references to events that resemble DS in a variety of cultural groups, such as Haitians and Native Americans (Francis & Baker, 1999). Studies of patients with DS have now been published from numerous geographical locations incorporating diverse cultural environments, including India (Goyal, Kalita, & Misra, 2014; J.P. Lazarus et al., 2003; Patidar et al., 2013; Wadwekar et al., 2014), Pakistan (Gul & Ahmad, 2014), South America (Alessi et al., 2013; d'Alessio et al., 2006; LaFrance et al., 2012; Marchetti et al., 2008; 2009; Portuguese et al., 2007; Proença et al., 2011; Scévola et al., 2013; Silva et al., 2001), China (An et al., 2010; Ding et al., 2013; 2014; Li et al., 2014; Xu et al., 2014), Iran (e.g. Asadi-Pooya, Emami & Emami, 2014), eastern/central Europe (Awad & Softic, 2011; Bora et al., 2011; Hovorka et al., 2007) and southern Europe (Baillés et al., 2004; Mari et al., 2006; Mazza et al., 2009; Rodriguez-Urrutia et al., 2014).

These findings suggest that DS may be a universal psychological disorder (LaFrance, Baker, et al., 2013). However, as discussed by Martinez-Taboas et al. (2010), there may be cultural influences on aetiological factors or manifestations of the disorder. For example, two studies have noted subtle differences in clinical manifestations of DS between Caucasian and African American patients (Abubakr & Wambacq, 2013; Abubakr, Wambacq, & Goerres, 2013). Furthermore, there may also be cultural differences in how DS are interpreted and responded to.

### **1.3. Clinical presentation**

#### **1.3.1. Symptoms**

Many patients diagnosed with DS present with frequent seizures that occur on a daily or weekly basis (Bodde et al., 2007; 2012; Carton et al., 2003; Davis, 2004; Dworetzky et al., 2005; Duncan et al., 2011; Goyal et al., 2014; Karakis et al., 2014; J.P. Lazarus et al., 2003; O'Brien et al., 2015; Scheepers et al., 1994; Szaflarski & Szaflarski, 2004; Wolf et al., 2015), and significantly disrupt patients' functioning (Bodde et al., 2013; R. Thompson et al. 2009). Patients with DS seem to experience more frequent seizures (Al Marzooqi et al., 2004; Dworetzky et al., 2005; Jędrzejczak, Owczarek, & Majkowski, 1999; Lally et al., 2010; Szaflarski & Szaflarski, 2004; Wolf et al., 2015) and reduced global functioning in comparison to patients with ES (Scévola et al., 2013).

Symptoms of DS vary considerably between cases (Reuber et al., 2011; Sirven & Glosser, 1998), and can resemble any type of epileptic seizure (ES), including: frontal, temporal, myoclonic, generalised and absence seizures (Bauer & Elger, 2001; Boon & Williamson, 1993; Bowman, 1998; Fejerman, 2005; Saygi et al., 1992; Twamley & Bortz, 1999). There have been inconsistencies in reports of the most common symptoms of DS, although expert opinion (Reuber, 2009) suggests that tonic-clonic like seizure presentations are the most frequent (i.e. excessive limb, trunk and head movements). The current review indicated that the most common symptoms of DS are major or minor positive motor symptoms, such as shaking, trembling, thrashing and jerking (Abubakr et al., 2003; An et al., 2010; Bodde, Lazeron, et al., 2012; Cianci et al., 2011; d'Alessio et al., 2006; Goyal et al., 2014; Gröppel, Kapitany, & Baumgartner, 2000; Hovorka et al., 2007; Hubsch et al., 2011; Lancman et al., 1993; 1994; Mitchell et al., 2012; Moore & Baker, 1997; Reuber, Pukrop, Bauer, et al., 2003; Selwa et al., 2000; Silva et al., 2001), and unresponsiveness and/or reported loss of awareness (An et al., 2010; Bodde et al., 2007; Cianci et al., 2011; Lancman et al., 1993; 1994; J.P. Lazarus et al., 2003; Moore & Baker, 1997; Patidar et al., 2013; Reuber, Pukrop, Bauer, et al., 2003; Reuber et al., 2011; Selwa et al., 2000).

Ictal eye closure (An et al., 2010; Asadi-Pooya, Emami, & Emami, 2014; Cianci et al., 2011; Hovorka et al., 2007; Hubsch et al., 2011; Patidar et al., 2013), hyperventilation

(Cianci et al., 2011; J.P. Lazarus et al., 2003) and peri-ictal auras (Asadi-Pooya, Emami & Emami, 2014; Hovorka et al., 2007) were also reported relatively regularly. Symptoms can also include: non-verbal vocalisations, weeping, pelvic thrusting, stiffening, back-arching, drying of the mouth, elevated heart rate, and 'pins and needles' (Gates et al., 1985; Hovorka et al., 2007; Lesser, 1996; Mellers, 2009). Several authors have reported signs of peri-ictal autonomic arousal and/or anxiety in this patient group (Galimberti et al., 2003; Goldstein & Mellers, 2006; Moore and Baker, 1997).

Patients with DS frequently perceive their seizures to come 'out-of-the-blue', without any warning signs or triggers (Reuber et al., 2011), although some patients report prodromal symptoms (Stone & Carson, 2013). Nonetheless, when triggers can be identified, they are often those not consistently seen in ES, such as sounds, pain, physical activity, lights and being 'upset' or stressed (Benbadis, 2009; Benbadis & LaFrance, 2010; R.J. Brown et al., 2011; LaFrance, Baker, et al., 2013). Triggers involving stressful circumstances are said to be indicative of DS (Benbadis & LaFrance, 2010; LaFrance, Baker, et al., 2013), although they do not reliably distinguish patients with ES and DS because stress can also precipitate ES (Allendorfer & Szaflarski, 2014; Benbadis & LaFrance, 2010).

On average, both onset and termination of seizures are more gradual than in ES (Francis & Baker, 1999; Fritzsche et al., 2013; Mostacci et al., 2011; Reuber, 2008). The severity of symptoms can vary considerably during an attack (Hubsch et al., 2011), and some patients with DS present with a variety of seizure symptoms and types (Bowman, 1998; Galimberti et al., 2003; Silva et al., 2001).

### **1.3.2. Seizure types**

A number of investigators have attempted to categorise DS into discrete types of seizure. Early approaches to classifying DS were largely dichotomous, with seizures being seen as either 'hypermotor' or 'atonic' (Griffith & Szaflarski, 2010). LaFrance, Baker, and colleagues (2013) maintain that the most common seizure types are 'convulsive/thrashing' with unresponsiveness, and 'falling' with unresponsiveness. A wide range of classification schemes has now been reported, ranging considerably in

specificity. In accordance with the above authors, only two main subtypes have been distinguished by some investigators (e.g. Abubakr et al., 2003), although others have distinguished between three or more (e.g. Bora et al., 2011; Dhiman, Sinha, Rawat, Harish, et al., 2013; N.M. Griffith et al., 2007; Gröppel et al., 2000; Hubsch et al., 2011; Selwa et al., 2000; Seneviratne, Reutens, & D'Souza, 2010). Whilst there were methodological weaknesses in many studies (e.g. mixed ES and DS included, inconsistent/unspecified seizure rating procedures), the general pattern in the literature is that of four main DS 'subtypes', including major motor, minor motor, unresponsiveness, and subjective/sensory symptoms. However, it is important to recognise that many patients do not fit into these discreet categories.

### **1.3.3. Prolonged DS**

Some patients present with 'pseudostatus', referring to seizures that are prolonged and often mistaken for status epilepticus (Dworetzky, Bubrick, & Szaflarski, 2010; S.J. Howell, Owen, & Chadwick, 1989; Rechin, Loew, & Joraschky, 1997). The proportion of DS patients who have experienced pseudostatus generally ranges from around 10-25% (Asadi-Pooya, Emami, Emami, & Sperling, 2014; Dworetzky et al., 2006; Hubsch et al., 2011), although Reuber, Pukrop, Mitchell, et al. (2003) reported a rate as high as 77.6% in one sample. The Non-epileptic Seizure Task Force (American Epilepsy Society) carried out a survey of US neurologists (Dworetzky et al., 2010) and found that 45% of them did not typically differentiate prolonged seizures from other types of DS. There also seems to be little in the way of standardised interventions for 'pseudostatus'. Recognition, treatment and management of 'pseudostatus' in patients with DS is an important area for further research, not least because inappropriate emergency medical treatment can be harmful, if not fatal (Reuber, Baker, et al., 2004).

### **1.3.4. Comorbidity of DS and ES**

Between 5 and 56% of patients with DS have been reported to have comorbid epilepsy (Asadi-Pooya & Emami, 2013; Benbadis, Agrawal, & Tatum, 2001; Bodde et al., 2012; 2013; Bora et al., 2011; Devinsky et al., 1996; Gambini et al., 2014; Hoepner et al., 2014; Hubsch et al., 2011; Lempert & Schmidt, 1990; Marchetti et al., 2009; Martin et al., 2003; Muller et al., 2002; O'Sullivan et al., 2007; Patidar et al., 2013; Pillai

& Haut, 2012; Scheepers et al., 1994; Silva et al., 2001). In patients with comorbid ES and DS, the onset of the epileptic disorder often occurs before the onset of DS (Devinsky et al., 1996; Magauidda et al., 2011). Moreover, the occurrence of individual ES and DS can occur in close temporal proximity (Andrade, Singh, & Bhakta, 2006; Devinsky & Gordon, 1998). Interestingly, some patients have been found to develop DS after epilepsy surgery (Markoula et al., 2013; Ney et al., 1998). On the other hand, one study showed that, in patients with pre-existing mixed ES/DS, the DS actually improved or resolved following neurosurgical interventions for epilepsy (Reuber, Kurthen, et al., 2002).

### **1.3.5. Clinical differences between ES and DS**

Whilst there is some overlap in symptoms, the presentation of DS differs from ES in several ways. DS tend not to conform to the usual stereotyped configurations of symptoms that are indicative of epilepsy (Benbadis, 2009; Boon & Williamson, 1993; Devinsky, Gazzola, & LaFrance, 2011; Goldstein & Mellers, 2012; Mellers, 2009). DS symptoms also vary throughout ('waxing and waning') and between seizures (Devinsky et al., 2011; Francis & Baker, 1999; Mellers, 2009).

A number of reviews have discussed the semiological characteristics that best discriminate ES and DS. Reviewers have generally found support for the following distinguishing characteristics of DS: asynchronous ('out-of-phase') movements, side-to-side head and/or body movements, fluctuating course/symptoms, pelvic thrusting, ictal eye closure, memory recall of ictal events, longer duration, and ictal weeping (Avbersek & Sisodiya, 2011; Devinsky et al., 2011; Gates, 2002; Goldstein & Mellers, 2012; Oto & Reuber, 2014; Mostacci et al., 2011). However, some of these symptoms can also be observed in frontal lobe seizures and so are of limited utility in distinguishing these from DS (e.g. Goldstein & Mellers, 2012).

Some studies have indicated that responsiveness/awareness is preserved more commonly in DS than in ES (Ali et al., 2010; W.L. Bell et al., 1998; M.C. Brown et al., 1991; Francis & Baker, 1999; Mostacci et al., 2011; Reuber & Kurthen, 2011; Syed et al., 2011). Furthermore, the following symptoms have also been reported to be more common in DS relative to ES: pre-ictal movements (D.B. Moore et al., 1998), ictal

stuttering (Vossler et al., 2004), ictal eye fluttering (Syed et al., 2011), post-ictal 'whispering', and partial motor responses to commands (Chabolla & Shih, 2006). On the other hand, DS are less likely to be accompanied by self-inflicted injury (e.g. tongue-biting) or incontinence (urinary/faecal), and occur less often at night or from sleep, relative to ES (Bazil & Walczak, 1997; M.C. Brown et al., 1991; French, 1995; Pegeuro et al., 1995). Some post-ictal symptoms also seem to be less common after DS than ES, such as headache and fatigue (Ettinger, Weisbrot, et al., 1999). Post-ictal breathing patterns may also be useful in distinguishing between DS and ES (Azar et al., 2008; Rosemergy et al., 2013; Sen, Scott, & Sisodiya, 2007).

Important methodological limitations in studies of the semiology of ES and DS were noted by Avbersek and Sisodiya (2011), such as a lack of control groups in some studies, inadequate definition of the signs under scrutiny, raters not being blind to diagnosis, and failure to differentiate between different types of ES and DS (i.e. convulsive, atonic, absences). Future studies might seek to address some of these limitations.

### **1.3.6. Quality of life**

Patients with chronic seizure disorders are generally faced with a number of challenges, including psychological difficulties, psychosocial problems, and physical symptoms that may restrict and impinge on a patient's well-being and lifestyle (Szaflarski & Szaflarski, 2004). As such, the symptoms of DS have a considerable impact on those who experience the disorder. Losses in social independence and reduced occupational functioning are often reported (Binder & Salinsky, 2007; Reuber, Howlett, et al., 2007; Scheepers et al., 1994; Twamley & Bortz, 1999), and patients with DS often receive some form of state financial assistance (Krahn et al., 1997; Moore & Baker, 1997). There is considerable debate about whether or not patients with DS should be permitted to drive, with many specialists recommending that they do not (Benbadis, Blustein, & Sunstad, 2000; Morrison & Razvi, 2011; Specht & Thorbecke, 2009).

Studies have shown that patients with DS experience reduced quality of life (QoL) when compared to control groups (Al Marzooqi et al., 2004; Breier et al., 1998;



Cronje & Pretorius, 2013; Hopp et al., 2012; Karakis et al., 2014; LaFrance et al., 2011; Strutt et al., 2011b; Szaflarski et al., 2003; Testa et al., 2007; van Merode et al., 2004; Wolf et al., 2015). Reduced QoL in DS seems to be related to the regularity of the attacks (Lawton et al., 2009), and only seems to improve after total cessation of seizures (Quigg et al., 2002). Moreover, QoL has been reported to be predicted by symptoms of depression and dissociation in patients with DS (Mitchell et al., 2012), indicating the importance of addressing such symptoms within psychological interventions for DS.

#### **1.4. Diagnosis**

A positive diagnosis of DS requires all other possible paroxysmal physiological and psychological phenomena to be excluded. One of the most important differential diagnoses is that of ES. In addition, other organic differential diagnoses include syncope, transient ischaemic attacks, migraines, substance-related events, hypoglycaemia, and sleep disorders (Benbadis & LaFrance, 2010; Duncan et al., 2011; Fritzsche et al., 2013; Griffith & Szaflarski, 2010; Mellers, 2005; 2009; Mihaescu & Malow, 2003; Petkar et al., 2006; Vossler et al., 1995). Psychological/psychiatric differential diagnoses include anxiety/panic attacks, psychosis, depersonalisation disorder, factitious disorder, and malingering (Alper et al., 1995; Mellers, 2005; 2009).

It often takes a considerable period of time for patients with DS to be referred to a specialist service for diagnostic investigation (Binder & Salinsky, 2007), although a subgroup of patients with a more significant history of psychological and medical complaints/interventions seem to be referred sooner (Bodde et al., 2012). Nonetheless, there is generally a considerable delay in reaching a diagnosis of DS (Jones et al., 2010; Seneviratne et al., 2011), with the average delay to diagnosis being approximately seven years (Alessi & Valente, 2013; Bodde et al., 2007; Dhiman, Sinha, Rawat, Harish, et al., 2013; Reuber, Fernández, Bauer, Helmstaedter, & Elger, 2002). The previous use of more AEDs and limited social support predict longer delays to diagnosis of DS (Rodriguez-Urrutia et al., 2014).

#### **1.4.1. Misdiagnosis of ES and DS**

Misdiagnosis of ES in patients with DS is a fairly common occurrence (Benbadis, 2005b; 2009; Bowman, 1998; Devinsky et al., 2011; Dhiman, Sinha, Rawat, Harish, et al., 2013; Hubsch et al., 2011; Krahn et al., 1997; Mellers, 2005; Moore & Baker, 1997; Oto & Reuber, 2014; Parra, Iriarte, & Kanner, 1999). However, due to increasing awareness and recognition of DS over time, patients with epilepsy may also be misdiagnosed with the former (Parra et al., 1999).

The proportion of patients with DS taking AEDs ranges from 44 to 100% (Benbadis, 1999; Bora et al., 2011; Bowman, 1993; Dworetzky et al., 2005; LaFrance & Syc, 2009; Lempert & Schmidt, 1990; Mitchell et al., 2012; Moore & Baker, 1997; Patidar et al., 2013; Reuber, Pukrop, Bauer, Helmstaedter, et al., 2003). Patients with DS may continue to be prescribed AEDs for prolonged periods of time (Bowman, 1998; Carton et al., 2003; Jones et al., 2010). Interestingly, some individuals with DS report reductions or cessation of DS whilst using AEDs, which presumably reflects a placebo effect (Alessi & Valente, 2014). Nevertheless, AED use entails considerable iatrogenic risk, including toxicity due to polypharmacy and possible teratogenic effects in female patients of child-bearing age (Francis & Baker, 1999; L.B. Holmes, Harvey, et al., 2001; Meador et al., 2009; Oto & Reuber, 2014; Vinten et al., 2005).

#### **1.4.2. Clinical assessment and investigations**

Patients with suspected DS often undergo numerous clinical assessments and investigations to determine the correct diagnosis (e.g. Ahmedani et al., 2013). Clinical history is usually evaluated, including previous/current symptoms, onset and course of the disorder (Griffith & Szaflarski, 2010; Sirven & Glosser, 1998). Descriptions of typical seizures are routinely sought from patients and a third party, and direct observation of an event is considered highly valuable (Mellers, 2009). Standardised self- and witness-rated questionnaires measuring clinical symptoms can be useful in indicating possible DS (Azar et al., 2010; Syed et al., 2009), as they provide a means of quantifying the attacks systematically. However, it is recommended that no single clinical sign should be relied upon during diagnostic evaluation (Alsaadi & Marquez, 2005; Avbersek & Sisodiya, 2010; Boon & Williamson, 1993; Devinsky et al., 2011; Goldstein & Mellers, 2012; LaFrance, Baker, et al., 2013). Information regarding

patients' social circumstances, psychosocial history, current emotional/mental functioning and psychiatric history may also be obtained (Francis & Baker, 1999; Schmutz, 2013).

Routine electroencephalography (EEG) is often used to determine the presence/absence of abnormal electrophysiological activity inter-ictally and/or ictally. Patients with DS generally show a lack of ictal epileptiform abnormalities on EEG output (Davis, 2004; Hovorka et al., 2007). However, EEG recordings may at times lead to misdiagnosis due to the fact that abnormal EEG activity unrelated to epilepsy can also occur in individuals who do not have epilepsy, including those with DS (e.g. Reuber, Fernández, Bauer, Singh & Elger, 2002). Movement artefact is another possible confounding issue when assessing EEG recordings during seizures (Bowman, 1998; Burnstine, Lesser, & Cole, 1991; Vinton et al., 2004).

Video-EEG can be used to monitor electrophysiological brain discharges whilst simultaneously recording patients' typical ictal behaviour on camera. This technique is usually carried out in epilepsy monitoring units on an inpatient basis, although outpatient video-EEG may be more appropriate in some cases (Benbadis et al., 2004; Devinsky et al., 2011; McGonigal et al., 2002; Seneviratne et al., 2012). Video-EEG is considered the 'gold-standard' in differential diagnostic procedures for DS (Eddy & Cavanna, 2014; French, 1995; LaFrance & Plioplys, 2012; LaFrance & Devinsky, 2004; LaFrance, Reuber & Goldstein, 2013; Mellers, 2009; Oto & Reuber, 2014; Sirven & Glosser, 1998). However, this method also has its limitations (Benbadis, 2009; Cragar et al., 2002).

Seizure provocation techniques are sometimes used to increase the likelihood of capturing a typical attack during the monitoring process (Schachter, Brown, & Rowan, 1996; Stagno & Smith, 1996). These techniques include: injection of a placebo solution, application of a tuning fork to the upper head, hyperventilation, application of a moist swab to the temples/back of neck, olfactory stimulation, temple pressure, 'head-up tilting', photo-stimulation, and hypnosis; some of which are presented with the verbal suggestion that the techniques are likely to induce a seizure (J.J. Barry, Atzman, & Morrell, 2000; Bazil et al., 1994; Benbadis, Johnson, et al., 2000; Bhatia et

al., 1997; Chen et al., 2011; Dericioglu, Saygi, & Ciger, 1999; Flugel et al., 1996; Goyal et al., 2014; Hoepner et al., 2013; Hovorka et al., 2007; Jędrzejczak et al., 1999; Khan et al., 2009; Kuyk et al., 1995; Kuyk, Spinhoven, & van Dyke, 1999; Lancman et al., 1994; Martinez-Taboas, 2002; McGonigal et al., 2002; Ney, Zimmerman, & Schaul, 1996; Ribai, Tugendhaft, & Legros, 2006; Schwarz, Bickford, & Rasmussen, 1955; Slater et al., 1995; Stagno & Smith, 1996; Staudenmeyer & Kramer, 1999; Wassmer, Wassmer, & Donati, 2003; Zaidi et al., 1999; Zalsman & Dror, 2002).

One study suggested better long-term outcomes in patients who had undergone seizure induction at diagnosis (Gambini et al., 2014); however, these techniques have been debated extensively on ethical grounds as deception is often necessary (Devinsky & Fisher, 1996; Gates, 2001; Goyal et al., 2014; Iriarte et al., 2003; Kuyk et al., 1997; Leeman, 2009; Schachter et al., 1996; M.L. Smith et al., 1997; Stagno & Smith, 1996; Updyke & Duryea, 2013; Whitaker & Rosenberg, 2001). Measurement of serum prolactin, cortisol, and other hormones (e.g. growth hormone, thyrotropin) post-ictally can also be useful in distinguishing ES and DS, as levels of these hormones are found to be elevated following ES but not DS (Gates, 2002; Kuyk et al., 1997; Rao, Stefan, & Bauer, 1989).

#### **1.4.3. Behavioural signs**

Some behavioural signs can be used to assist in discriminating between patients with DS and ES. For example, having a seizure in a physician's office or in the presence of an audience is considered to be indicative of DS as opposed to ES (Benbadis, 2005a). The 'teddy bear sign' is another interesting behavioural indicator of DS. Burneo et al. (2003) noted that significantly more patients with DS brought an age-inappropriate soft-toy to the monitoring unit than patients with ES. Indeed, Hoerth and colleagues (2008) identified the 'teddy bear sign' as one of the strongest predictors of a DS diagnosis. However, the teddy bear sign is low in sensitivity due to relatively infrequent occurrence, and it is less useful in patients over 18 years old (Cervenka et al., 2013).

Linguistic analysis has also been used to distinguish between patients with DS and ES, with considerable success (Cornaggia et al., 2012; Plug, Sharrack, & Reuber, 2009; Reuber et al., 2009; Reuber & Plug, 2009; Robson et al., 2012; Schwabe, Howell, &

Reuber, 2007). When asked to describe their typical attacks, patients with DS tend to respond in characteristic ways, such as showing resistance to answering specific questions, reporting amnesia for the events, conceptualising them as phenomena arising internally, confabulation/reconstruction of events, and catastrophisation in descriptions of their disorder.

#### **1.4.5. Difficulties in the diagnosis of DS**

Once confirmed, a positive diagnosis of DS can lead to a number of beneficial outcomes, including improvements in seizure frequency (Farias, Thieman, & Alsaadi, 2003; Martin et al., 1998; Mayor et al., 2012) and reduced healthcare utilisation (Ahmedani et al., 2013; Jirsch et al., 2011; Martin et al., 1998; McKenzie et al., 2010; Razvi, Mulhern, & Duncan, 2011). Nonetheless, patients can respond to the diagnosis with a variety of emotions including relief, anger and anxiety (Duncan, Graham, & Oto, 2014a; Ettinger, Dhoon, et al., 1999; Karterud, Knizek, & Nakken, 2010; Riaz et al., 1998; Scheepers et al., 1994).

Patients can be left confused by the diagnosis (Carton et al., 2003; Dickinson, Looper, & Groleau, 2011; Green, Payne, & Barnitt, 2004; R. Thompson et al., 2009; Wyatt, Laraway, & Weatherhead, 2014), and may feel concerned that their symptoms are 'all in their head' or that they are 'crazy' (Brown & Trimble, 2000; N.C. Thompson, Osorio, & Hunter, 2005, p.75). Moreover, patients may interpret the diagnosis as implying deliberate feigning of symptoms (Karterud et al., 2010; N.C. Thompson, Osorio, & Hunter, 2005). There can be fear of possible stigma related to a functional diagnosis, or concerns about not being believed by medical professionals (Green et al., 2004; Karterud et al., 2010; Wyatt et al., 2014). These concerns may not be entirely unfounded, as some relevant healthcare professionals (e.g. inpatient nurses, psychiatrists, neurologists, general practitioners, emergency medicine physicians) seem to view DS as within the voluntary control of the patients to some extent (Baslet, 2012; Sahaya et al., 2012; Shneker & Elliott, 2008; Whitehead, Kandler, & Reuber, 2013; Whitehead & Reuber, 2011; Worsley et al., 2011).

Some factors, however, seem to facilitate coping with a diagnosis of DS. These include achieving a good understanding of possible psychological mechanisms

underlying the seizures, possessing an internal locus of control, receiving respectful treatment by healthcare professionals, meeting others with the disorder, and being given a clear indication as to what to expect in terms of follow-up and treatment (Dickinson et al., 2011; Karterud et al., 2010). A number of protocols have now been developed to standardise and improve the way in which diagnoses of DS are presented to patients (Duncan, 2010; Hall-Patch et al., 2010; Mellers, 2009; Shen, Bowman, & Markand, 1990). Recently, guidelines have been published, providing a staged approach to the diagnosis of DS (LaFrance, Baker, et al., 2013), with four levels of diagnostic certainty that can be achieved depending on the data available.

### **1.5. Treatment**

Many patients with DS are treated with 'standard medical care', involving presentation of the diagnosis, gradual withdrawal of AEDs, follow-up with a neurologist, and/or referral to psychiatry or psychology (LaFrance, Rusch, & Machan, 2008). Confirming and presenting the diagnosis of DS to patients is widely seen as the initial step in treating the disorder (Baslet, 2012; Brown & Trimble, 2000; LaFrance, Reuber, et al., 2013; Lesser, 2003). Simply receiving the diagnosis can bring about improvements in symptoms for some patients (Farias et al., 2003; Gambini et al., 2014; Lesser, 1996; Oto & Reuber, 2014).

Psychological treatment approaches in the literature suggest a range of possibly beneficial approaches, including cognitive behavioural therapy (CBT; Conwill et al., 2014; Goldstein et al., 2004; 2009; 2010; Kuyk et al., 2008; LaFrance et al., 2009; 2014), psychodynamic psychotherapy (Reuber, Burness, et al., 2007), psychoeducation (Baxter et al., 2012; Mayor et al., 2013; N.C. Thompson et al., 2013), group psychotherapy (J.J. Barry et al., 2008; Conwill et al., 2014; Metin et al., 2013; Prigatano, Stonnington, & Fisher, 2002; Zaroff et al., 2004), behavioural techniques (DeLeon, Uy, & Gutshall, 2005), hypnotherapy (H.R. Miller, 1983; Moene et al., 2002), mindfulness-focused approaches (Baslet & Hill, 2011; Baslet et al., 2014), paradoxical intention therapy (Ataoglu et al., 2003; Chapleau et al., 2013) eye movement desensitisation and reprocessing (EMDR; Chemali & Meadows, 2004; Kelley & Benbadis, 2007; Kelley, Benbadis, & Adams, 2005), and integrated therapies (Howlett & Reuber, 2009).

Some biological interventions have also been explored. The use of selective serotonin reuptake inhibitors (SSRIs; e.g. sertraline, venlafaxine) have been associated with positive results in two studies (LaFrance et al., 2010; Pintor et al., 2010). Moreover, biofeedback (Swingle, 1998) and electroconvulsive shock therapy (Blumer, Rice, & Adamolekun, 2009) have also been used to treat DS, although the known risks and side-effects associated with the latter should contraindicate its use. Research has also started to explore the efficacy of transcranial magnetic stimulation in patients with conversion disorders including those with DS (Parain & Chastain, 2014), although the possible risks of this technique should also be considered.

At present, empirically sound research into the efficacy of treatments for DS is fairly sparse, with only a few published controlled trials. A Cochrane Review of controlled treatment trials in this patient group (Baker et al., 2007; Brooks et al., 2007) identified only three studies meeting the inclusion criteria of either full- or quasi-randomisation to treatment groups. Nevertheless, the situation is gradually improving. For example, a multi-centre randomised control trial of CBT (with or without sertraline) has recently been published in the US (LaFrance et al., 2014), and another is in progress in the United Kingdom (Goldstein et al., in progress; ISRCTN05681227). However, there are a number of challenges faced by patients, clinicians and investigators in receiving, providing and evaluating therapeutic interventions for DS respectively. These include treatment adherence, attitudes and expectations of patients and clinicians, secondary gain from illness roles, drug interactions, frequent seizures leading to poor attendance at appointments, availability and funding, to name just a few.

### **1.6. Outcomes**

Studies have shown that outcomes have generally been poor to moderate in this patient group (LaFrance & Devinsky, 2002), with only a proportion of patients showing seizure remission or improvements at follow-up. Rates of seizure cessation have varied between 17-55% (Aboukasm et al., 1998; An et al., 2010; Arain et al., 2007; Betts & Boden, 1992a; Bodde et al., 2007; Duncan et al., 2011; Duncan, Graham, & Oto, 2014b; Ettinger, Devinsky, et al., 1999; Ettinger, Dhoon et al., 1999; Hovorka

et al., 2007; Jones et al., 2010; Jongsma et al., 1999; Kanner et al., 1999; Krahn et al., 1997; Kristensen & Alving, 1992; Lempert & Schmidt, 1990; McKenzie et al., 2010; Patidar et al., 2013; Reuber, Pukrop, Bauer, et al., 2003; Riaz et al., 1998; Selwa et al., 2000; Silva et al., 2001; Walczak et al., 1995), although higher rates have been reported on occasion (e.g. Gambini et al., 2014). However, patients who continue to have seizures at follow-up often report lower frequencies and/or reduced severity, compared to baseline (Aboukasm et al., 1998; Betts & Boden, 1992a; Bodde et al., 2007; Buchanan & Snars, 1993; Duncan, Graham, & Oto, 2014b; Ettinger Devinsky, et al., 1999; Ettinger, Weisbrot et al., 1999; Gambini et al., 2014; Hovorka et al., 2007; Krahn et al., 1997; Patidar et al., 2013; Riaz et al., 1998; Selwa et al., 2000; N.C. Thompson, Osorio, & Hunter, 2005; Walczak et al., 1995; Zhang et al., 2009).

Poorer outcomes have been found to be associated with a number of variables including comorbid psychiatric diagnoses and psychopathology at baseline (Bodde et al., 2007; Durrant, Rickards, & Cavanna, 2011; Kanner et al., 1999; Lempert & Schmidt, 1990; McKenzie et al., 2010; Reuber et al., 2003; Walczak et al., 1995), longer duration of disorder (Lempert & Schmidt, 1990; Selwa et al., 2000; Walczak et al., 1995), worse reported occupational functioning (Duncan et al., 2011; Ettinger, Devinsky, et al., 1999), receiving social security benefits (McKenzie et al., 2010), later age at seizure onset/diagnosis (An et al., 2010; Reuber et al., 2003), more negativism (Bodde et al., 2007), attending the first clinic visit unaccompanied (Arain et al., 2007) and more 'dramatic' or motor ictal symptoms (Arain et al., 2007; Betts & Boden, 1992a; Durrant et al., 2011; Reuber et al., 2003; Selwa et al., 2000).

Furthermore, female gender (McKenzie et al., 2010), comorbid ES (Durrant et al., 2011), lower educational attainment (Arain et al., 2007; Reuber et al., 2003), a below average or lower intelligence quotient (IQ) score (McDade & Brown, 1992), worse perceived general health (Ettinger, Devinsky, et al., 1999), atypical magnetic resonance imaging (MRI) results (Kanner et al., 1999), less positive current/childhood relationships (Durrant et al., 2011; Ettinger, Dhoon, et al., 1999) and a history of violent behaviour (McDade & Brown, 1992), have also been linked to worse outcomes. On the other hand, acceptance of the diagnosis by patients and caregivers



seems to be associated with more positive outcomes (Duncan, Graham, & Oto, 2014a; Ettinger, Devinsky, et al., 1999).

### **1.7. Summary and conclusions**

Dissociative seizures (DS) are transient episodic events that superficially resemble epilepsy, but have no clear organic basis. Patients present with a wide variety of symptoms, which vary within and between individuals with the disorder. Differential diagnosis is complex and often considerably delayed, and can include numerous diagnostic tests. At present, within a limited evidence base, the best documented treatments include CBT, psychoeducation and integrated psychotherapies. The currently available research indicates variable outcomes, with many patients continuing to have seizures, reduced quality of life and poor psychosocial functioning, despite having received the diagnosis and/or interventions.

## **Chapter 2. The aetiology of dissociative seizures**

Contemporary perspectives on the aetiology of DS (e.g. Bodde et al., 2009; Mellers, 2009; Oto & Reuber, 2014; Reuber, 2009) incorporate a wide range of environmental / social, psychological and biological factors, variously conceptualised as predisposing, precipitating, and perpetuating variables. Predisposing variables are those typically present during development, which increase the risk of the development of the disorder at some later stage. Precipitating factors occur in close proximity to the onset of the disorder and are thought to directly trigger the first occurrence of symptoms. Perpetuating variables are those factors serving to maintain the disorder, once initiated. Some authors (e.g. Bodde et al., 2009; Reuber, 2009) further differentiate 'shaping' factors (factors influencing the development of seizures rather than a different type of symptom) and/or triggering factors (those contributing to the initiation of individual seizures).

The current chapter presents an overview of the empirical literature on the possible aetiological factors in DS. The review is based on the literature search described in Chapter 1. Only literature referring to variables of direct relevance to the current thesis is discussed in detail. The chapter begins with biological factors, continues with social / environmental variables, before summarising findings regarding psychological variables in patients with DS. The chapter then provides an outline of theoretical perspectives on the disorder. The overall aim of the chapter is, therefore, to give a general account of the aetiology of DS. Literature concerning dissociative and emotional processes in DS is discussed in Chapter 3.

### **2.1. Biological factors**

#### **2.1.1. Medical comorbidity and correlates**

Many patients with DS report multiple health symptoms and some have considerable medical histories, including neurological conditions, suspected or actual cancer, asthma, obesity, diabetes, gastro-oesophageal disease, allergies, ulcers and cardiovascular disease / hypertension (deWet et al., 2003; Elliott & Charyton, 2014; Jiminez, Sharma,

& Dar, 2014; Lempert & Schmidt, 1990; Marquez et al., 2004; Al Marzooqi et al., 2004; Park et al., 2014; Tojek et al., 2000), although it is possible that some of these could be misdiagnosed functional symptoms (see section 2.3.2).

Nonetheless, DS have also been reported following invasive or risky medical procedures, such as general anaesthesia (Lichter et al., 2004; Ramos & Brull, 2013), and neurosurgical procedures (Alessi & Valente, 2013; Davies et al., 2000; Glosser, Roberts, & Glosser, 1999; Markoula et al., 2013; Ney et al., 1998; Parra et al., 1998; Reuber, Kral, et al., 2002). DS can occur during pregnancy, although most often these are not 'de novo' (Brady & Huff, 1997; Carlson & Caplan, 2011; DeToledo, Lowe, & Puig, 2000; Devireddy & Sharma, 2015; Jain et al., 2013).

Reuber (2009) categorised physical illness and minor surgical procedures with other adult life events that might serve as precipitating factors in DS. It is possible that pain, distress and trauma due to physical illness, for example, could act in the same way as other stressors in the disorder (see section 2.2.2. below), by elevating levels of emotional distress or arousal to intolerable levels and thereby triggering the initial onset of the disorder. However, such experiences might also serve to predispose to the disorder (Oto & Reuber, 2014), perhaps by increasing attentional allocation to bodily experiences. On the other hand, physical illnesses/symptoms might also perpetuate DS, by contributing to ongoing psychological distress.

### **2.1.2. Head injury**

A history of reported head injury is also relatively common in patients with DS, although rates range substantially from 4-100% (Ahmedani et al., 2013; An et al., 2010; Asadi-Pooya, Emami & Emami, 2014; E. Barry et al., 1998; Bowman, 1993; Elliott & Charyton, 2014; LaFrance & Syc, 2009; LaFrance, DeLuca, et al., 2013; Lancman et al., 1993; Mökleby et al., 2002; Reuber et al., 2011; Salinsky et al., 2011; Scheepers et al., 1994; Snyder et al., 1994; Westbrook, Devinsky, & Geocadin, 1998), and reported head injuries tend to be minor in this group (E. Barry et al., 1998; LaFrance, DeLuca, et al., 2013; Westbrook et al., 1998). Rates of head injury have been found to be higher in patients with DS relative to those with somatisation disorder, healthy controls (Mökleby et al., 2002), and those with ES (Elliott & Charyton, 2014; Locke et al., 2006).

Indeed, a history of head injury has been found to be predictive of a diagnosis of DS rather than ES (Elliott & Charyton, 2014).

The wide variation in rates of head injury reported in previous studies could be due to methodological differences, such as the inclusion of patients with possible/confirmed comorbid ES in some instances (e.g. LaFrance & Syc, 2009; Mökleby et al., 2002). Moreover, only a few studies mentioned whether loss of consciousness occurred during the head injury (e.g. Lancman et al., 1993; Locke et al., 2006; Reuber et al., 2011), or whether the reported head injuries were antecedent to the onset of DS or not (e.g. An et al., 2010; LaFrance, DeLuca, et al., 2013). Nonetheless, it is possible that head injury could act both as a predisposing and precipitating factor for DS (Bodde et al., 2009; Reuber, 2009).

### **2.1.3. Neuroimaging**

#### ***Structural***

Patients with DS are more likely to have normal structural neuroimaging results than patients with ES (Drane et al., 2006; Dworetzky et al., 2005; Karakis et al., 2014; Locke et al., 2006; Rotge et al., 2009; Szaflarski et al., 2003; Szaflarski & Szaflarski, 2004). However, structural MRI abnormalities have been found in patients with DS, with rates of 18-65% (Devinsky, Mesad, & Alper, 2001; Hovorka et al., 2007; Jones et al., 2010; Reuber, Fernández, Helmstaedter, Qurishi, & Elger, 2002). Abnormalities reported include Arnold-Chiari malformation, (sub)arachnoid cysts, gliosis, neurosurgical lesions, generalised atrophy, changes characteristic of multiple sclerosis and medial temporal sclerosis (Benbadis, Tatum, et al., 2000; Hovorka et al., 2007; Reuber et al., 2002), and are most often noted in the right hemisphere (Devinsky et al., 2001). However, the elevated rates of MRI abnormalities could be linked to the inclusion of patients with comorbid ES in some studies (e.g. Devinsky et al., 2001; LaFrance & Syc, 2009). Nevertheless, abnormal findings have also been reported in DS patients without comorbid ES (e.g. Hovorka et al., 2007; Jones et al., 2010; Reuber, Fernández, Helmstaedter, Qurishi, & Elger, 2002).

Advanced volumetric analyses (i.e. voxel based morphometry, cortical thickness analysis) have recently revealed possible changes in neural matter in regions associated

with motor control, command and integration (right hemisphere motor and premotor areas, bilateral cerebellum), relative to healthy controls (Labate et al., 2012). However, there was no mention of patients' current/previous use of AEDs in this report. It is known that phenytoin, for example, is associated with cerebellar atrophy (e.g. de Marco et al., 2003; Ney et al., 1994), and so, exposure to such medications may have influenced the results of the study.

### **Functional**

Possible differences in functional neural activity during wakeful resting have been reported in patients with DS, relative to healthy controls. Hypometabolism has been observed in regions associated with emotional control (i.e. anterior cingulate cortex) and spatial awareness (i.e. parietal cortex), with positron emission tomography (Arthuis et al., 2014). Moreover, connectivity alterations have been reported in regions important for emotion, sensorimotor control, attention, executive functioning, and the 'default mode network' (i.e. neural regions usually active during wakeful rest), with functional MRI techniques (Ding et al., 2013; 2014; Li et al., 2014; van der Kruijs et al., 2012; 2014).

Interestingly, some of the connectivity differences were significantly correlated with scores on measures of dissociation in the DS patients in two studies (van der Kruijs et al., 2012; 2014). Moreover, connectivity values between the insula and supplementary motor area were positively correlated with seizure frequency in one study (Li et al., 2014). However, some patients included in the studies were currently (van der Kruijs et al., 2012) or had recently been (Ding et al., 2013; 2014; Li et al., 2014) taking medication including AEDs; therefore, this represents a possible confound. Furthermore, patients with ES and psychiatric comorbidity were excluded from these studies, thereby limiting the generalizability of the findings.

Further research is necessary in this area, particularly extending the functional neuroimaging research beyond resting state studies, to include studies utilising psychological tasks during scanning. Nonetheless, it seems that functional alterations in neural networks and non-specific structural abnormalities may be predisposing neurobiological factors in this patient group, although these relationships are as yet

poorly understood (Reuber, 2009).

#### **2.1.4. Electrophysiology**

Patients with DS are less likely to show abnormalities on EEG output than patients with ES (Ali et al., 2010; Drane et al., 2006; Karakis et al., 2014; Locke et al., 2006; Rotge et al., 2009; Schramke et al., 2007). Nevertheless, non-specific and/or inter-ictal EEG abnormalities are frequently found in patients with DS (Jawad et al., 1995; Lelliott & Fenwick, 1991; Snyder et al., 1994; Xue et al., 2013); with around 40-50% of patients showing such signs (Hovorka et al., 2007; Mökleby et al., 2002; Reuber, Fernández, Bauer, Singh, & Elger, 2002). Resting state spatial EEG patterns have recently been used to discriminate patients with ES and DS, with 92% accuracy (Xu et al., 2014).

In addition, a proportion of patients with DS exhibit epileptiform EEG changes (Reuber, Fernández, Bauer, Singh, & Elger, 2002). Recently, intracranial recording detected altered activity in the parietal cortex of a patient during a DS, despite a lack of overall EEG alteration associated with the seizure (Arzy et al., 2014). Moreover, Krishnan et al. (2011) observed that the gradual electrophysiological ‘entrainment’ that is typically observed leading up to seizure occurrence was more likely to be ‘reset’ after an ES than after DS. In fact, no such resetting occurred in the patients who experienced DS. Together, these findings indicate subtle peri-ictal electrophysiological differences in individuals with DS, although the meaning of these findings is as yet unclear.

Some EEG studies have examined electrophysiological changes after the presentation of specific stimuli in patients with DS. For example, more rapid auditory event-related potentials (ERPs; Drake et al., 1993), and altered frontocentral ERPs during a ‘mismatch negativity’ paradigm (Gene-Cos et al., 2005) have been reported in patients with DS (relative to those with ES and healthy controls respectively). Furthermore, reduced ‘sensory gating’ in patients with DS (relative to healthy controls) has also been observed in two studies (Almis et al., 2013; Pouretmad et al., 1998), suggestive of reduced automatic filtering of irrelevant sensory information.

The observed electrophysiological differences in DS could be linked to the structural

and functional brain abnormalities described previously. Such differences could have emerged during development, or might be a result of minor head injuries and comorbid neurological illness in this group. As such, these differences may interact in predisposing an individual to developing DS.

#### **2.1.5. Autonomic nervous system (ANS) functioning**

Heart rate variability (HRV) refers to the degree of variation in the interval between successive heart beats. Low levels of HRV are associated with reduced activity of the vagus nerve and parasympathetic ANS, and thus increased ANS activation. Reduced HRV at rest, as measured by several parameters, has been observed in patients with DS relative to healthy controls (Bakvis, Roelofs, et al., 2009; Ponnusamy, Marques, & Reuber, 2011; Roberts et al., 2012). However, some investigators have failed to find such differences (Müngen, Berilgen, & Arıkanoglu, 2010).

Patients with DS showed no differences in HRV relative to those with ES, when at rest (Ponnusamy, et al., 2011). On the other hand, patients with DS show higher levels of peri-ictal HRV compared to those with ES (Müngen et al., 2010; Ponnusamy, Marques, & Reuber, 2012). Together, these findings suggest generally elevated levels of sympathetic arousal in patients with DS (relative to healthy individuals), which are reduced during and around the time of a DS. A speculative interpretation is that the DS somehow serves as a mechanism to reduce sympathetic arousal.

Findings regarding peri-ictal heart rate have been variable. Reinsberger et al. (2012) found that DS patients' heart rates were elevated pre-ictally and reduced post-ictally, compared to patients with ES. Moreover, Opherk and Hirsch (2002) reported that heart rate was significantly lower in DS relative to ES, both ictally and post-ictally. Again, this would suggest that autonomic arousal is somehow inhibited or reduced during and immediately after a DS.

Patients diagnosed with DS also display elevated basal cortisol levels relative to healthy control participants, when measured using salivary sampling methods (Bakvis, Spinhoven, Giltay, et al., 2010). This supports the notion that DS are associated with a state of ongoing elevated stress reactivity (HPA; hypothalamic-pituitary-adrenal axis).

Together, the available literature seems to indicate an overall pattern of elevated sympathetic ANS activation at rest in patients with DS, in addition to reduced sympathetic activation during and after seizure occurrence.

It is possible that generally elevated ANS activation could constitute a predisposing or precipitating factor, increasing the risk of the onset of the disorder. However, an acute elevation in ANS activation may also trigger individual seizures (Goldstein & Mellers, 2006). Moreover, reductions in ANS activation during and after a seizure might represent part of the dissociative mechanism that occurs during DS.

## **2.2. Social / environmental factors**

### **2.2.1. Traumatic experiences**

#### ***General trauma***

A systematic review of studies on traumatic experiences in DS was published by Fiszman and colleagues (2004). On the basis of 17 studies that included control groups and evidence of DS based on video-EEG, lifetime rates of general trauma were reported to vary between 76 and 100%. Traumatic experiences reported by patients with DS include abuse, experiencing/witnessing violence, bereavement(s), witnessing trauma/death, accidents, bullying, combat- or war-related experiences, and medical trauma (Baillés et al., 2004; Bowman & Markand, 1996; Chen & Izadyar, 2009; Duncan & Oto, 2008; Moore & Baker, 1997; Myers, Perrine, et al., 2013; Quinn et al., 2012; Reuber, Howlett, et al., 2007; Salinsky et al., 2012; Scévola et al., 2013; Snyder et al., 1994; Tojek et al., 2000; Turner et al., 2011).

Of the studies reviewed presently, the proportion of DS patients reporting at least one type of trauma varied widely, ranging between 10 and 100% (An et al., 2010; Arnold & Privitera, 1996; Bodde et al., 2013; Bora et al., 2011; Bowman, 1993; Bowman & Markand, 1996; Dikel, Fennell, & Gilmore, 2003; Duncan & Oto, 2008; Hingray et al., 2011; LaFrance & Syc, 2009; Myers et al., 2013; Reuber, Howlett, et al., 2007; Rosenberg et al., 2000; Scévola et al., 2013; Turner et al., 2011; Wolf et al., 2015), with over half of these studies citing rates of 70% or more. Reports of dual or multiple traumatic life



events are not uncommon, with rates ranging from 24-74% (Bodde et al., 2013; Bowman, 1993; Duncan & Oto, 2008; Myers et al., 2013).

Patients with DS report higher rates of overall lifetime and childhood trauma compared to control groups (Arnold & Privitera, 1996; Dikel et al., 2003; Ozcetin et al., 2009; Proença et al., 2011; Reuber, Howlett, et al., 2007; Rosenberg et al., 2000; Scévola et al., 2013; Turner et al., 2011; van Merode et al., 2004), although there have been exceptions (e.g. Lally et al., 2010; O'Brien et al., 2015). Moreover, trauma history is significantly predictive of a diagnosis of DS rather than ES, with specificity of 86% and sensitivity of 76% (Arnold & Privitera, 1996).

A history of trauma is associated with reduced quality of life in individuals with DS (Myers et al., 2012; Wolf et al., 2015). Patients with DS and a history of trauma are also more likely to report higher levels of general psychopathology, dissociation, and PTSD symptoms. In addition, they are also more likely to have additional psychiatric diagnoses (particularly affective or anxiety disorders), and a history of suicidal behaviour (Hingray et al. 2011; Lally et al., 2010; Myers et al., 2013). Duncan and Oto (2008) identified distinct predictors of different types of trauma in patients with DS (e.g. gender, age, comorbidities). These findings suggested subgroups of patients with potentially distinct aetiological factors contributing to the development of the disorder.

### ***Sexual abuse***

Rates of lifetime sexual abuse in patients with DS vary considerably in the literature, ranging from 0 to 80% (Alper et al., 1993; An et al., 2010; Arnold & Privitera, 1996; Asadi-Pooya, Emami & Emami, 2014; Baillés et al., 2004; Bakvis, Roelofs, et al., 2009; Bakvis, Spinhoven, Giltay, et al., 2010; Betts & Boden, 1992b; Binzer, Stone, & Sharpe, 2004; Bowman, 1993; Bowman & Markand, 1996; Duncan & Oto, 2008; Duncan et al., 2011; Ettinger, Dhoon, et al., 1999; Holman et al., 2008; Jawad et al., 1995; Koby et al., 2010; Kuyk, Spinhoven, Boas, & van Dyke, 1999; Lancman et al., 1993; Litwin & Cardeña, 2000; Moore & Baker, 1997; Myers et al., 2013; Reuber, Howlett, et al., 2007; Scévola et al., 2013; Scheepers et al., 1994; Schramke et al., 2010; Selkirk et al., 2008; Silva et al., 2001; Spinhoven et al., 2004; Stone, Sharpe, & Binzer, 2004; Strutt et al., 2011a,b;

Thomas et al., 2013). Only five studies reported rates below 10% (An et al., 2010; Arnold & Privitera, 1996; Asadi-Pooya, Emami & Emami, 2014; Jawad et al., 1995; Silva et al., 2001), and around 50% of studies reported proportions between 20 and 40% (Alper et al., 1993; Binzer et al., 2004; Duncan & Oto, 2008; Duncan et al., 2011; Ettinger, Dhoon, et al., 1999; Koby et al., 2010; Kuyk et al., 1999; Myers et al., 2013; Reuber et al., 2007; Scévola et al., 2013; Schramke et al., 2010; Selkirk et al., 2008; Spinhoven et al., 2004; Stone, Sharpe, & Binzer, 2004; Strutt et al., 2011a,b; Thomas et al., 2013). Of the studies reviewed here, the average rate of lifetime sexual abuse was approximately 30%.

Studies reporting childhood sexual abuse (CSA) separately suggest its occurrence in between 17 and 85% of patients (Akyuz et al., 2004; Bowman, 1993; Bowman & Markand, 1996; 1999; Dikel et al., 2003; Koby et al., 2010; Lally et al., 2010; McDade & Brown, 1992; Portuguez et al., 2007; Prigatano, Stonnington, & Fisher, 2002; Reilly et al., 1999; Salmon et al., 2003). Seventy-five percent of these studies reported rates of CSA between 30 and 75% (Akyuz et al., 2004; Bowman, 1993; Bowman & Markand, 1996; 1999; Dikel et al., 2003; Koby et al., 2010; Portuguez et al., 2007; Reilly et al., 1999; Salmon et al., 2003). Only two studies cited rates below 30% (Lally et al., 2010; McDade & Brown, 1992). The average rate of CSA across all studies was approximately 44%. In contrast, the reported rate of CSA in the general population is approximately 4.8% (Radford et al., 2011). A systematic review and meta-analysis of studies reporting rates of sexual abuse in patients with DS suggested an average rate of 35.7%, compared to 16.6% in comparison groups (Sharpe & Faye, 2006). Patients were nearly three times as likely as controls to have a history of CSA (Odds Ratio of 2.940).

Regarding sexual assault/rape in adulthood, the findings have been more consistent, ranging from 23 to 36% (Bowman, 1993; Bowman & Markand, 1996; Koby et al., 2010; Lally et al., 2010; Reilly et al., 1999; Salmon et al., 2003). The average rate of adulthood sexual trauma across these six studies was 29.7%. In comparison, statistics suggest that approximately 20% of women in the general population have experienced some form of sexual violence since the age of 16 years (Office for National Statistics, 2013).

Patients with DS have been found to report significantly higher rates of sexual abuse than patients with ES (Akyuz et al., 2004; Alper et al., 1993; Binzer et al., 2004; Dikel et al., 2003; Holman et al., 2008; Kuyk et al., 1999; Litwin & Cardeña, 2000; Salmon et al., 2003; Scévola et al., 2013; Schramke et al., 2010; Strutt et al., 2011a,b), healthy controls (Bakvis, Roelofs, et al., 2009; Bakvis, Spinhoven, Giltay, et al., 2010; Ozcetin et al., 2009), and those with conversion movement disorder (Driver-Dunckley et al., 2011; Stone, Sharpe, & Binzer, 2004). However, a number of studies showed no statistically significant differences between DS and comparison groups (Arnold & Privitera, 1996; Berkhoff et al., 1998; Jawad et al., 1995; Koby et al., 2010; Lally et al., 2010; Proença et al., 2011; Rosenberg et al., 2000), or did not include inferential statistics for the direct comparison of DS with another group (Betts & Boden, 1992b; Bowman & Markand, 1999; McDade & Brown, 1992; Reilly et al., 1999; Reuber, Howlett, et al., 2007; Spinhoven et al., 2004).

The duration of sexual abuse has been found to distinguish between patients with ES and those with DS, with longer duration significantly predicting the latter diagnosis (Litwin & Cardeña, 2000). Some studies have indicated that sexual abuse/assault is more common in female patients with DS (Bowman & Markand, 1996; Salmon et al., 2003; Selkirk et al., 2008; Thomas et al., 2013), which is possibly reflective of the general tendency for females to be at higher risk of abuse in general (DiTomasso & Routh, 1993).

A history of sexual abuse has been linked to a wide variety of characteristics in patients with DS, including a history of other mental health diagnoses, self-harm, diagnosis of personality disorder, other medically unexplained symptoms (MUS), more frequent emotional antecedents to seizures, the use of more than two AEDs, receiving state benefits, a history of physical abuse, younger age at seizure onset, longer time to diagnosis, previous referral to secondary care mental health services, convulsive / 'abreactive' DS, more severe DS, prodromal symptoms, flashbacks, nocturnal DS, self-injurious ictal symptoms, and ictal urinary incontinence (Betts & Boden, 1992b; Duncan & Oto, 2008; Selkirk et al., 2008).

In summary, it seems that sexual abuse, particularly during childhood, is more common

in patients with DS than a number of control groups and the general population. Furthermore, patients with DS who have a history of sexual abuse have poorer mental health and more severe/complex DS, compared to those who do not.

### ***Physical abuse***

Lifetime rates of physical abuse have also ranged enormously, from 1 to 100% across samples (Alper et al., 1993; An et al., 2010; Arnold & Privitera, 1996; Asadi-Pooya, Emami, & Emami, 2014; Baillés et al., 2004; Bakvis, Roelofs, et al., 2009; Bakvis, Spinhoven, Giltay, et al., 2010; Bowman, 1993; Bowman & Markand, 1996; 1999; Dikel et al., 2003; Driver-Dunckley et al., 2011; Duncan & Oto, 2008; Duncan et al., 2011; Ettinger, Dhoon, et al., 1999; Koby et al., 2010; Lancman et al., 1993; Litwin & Cardeña, 2000; Moore & Baker, 1997; Myers et al., 2013; Scévola et al., 2013; Selkirk et al., 2008; Spinhoven et al., 2004; Strutt et al., 2011a,b; Thomas et al., 2013). There were 26 studies reporting physical abuse rates without specifying an age range, with an average percentage of 37%.

Childhood physical abuse is reported by 6-80% of patients with DS (Akyuz et al., 2004; Bowman, 1993; Bowman & Markand, 1996; 1999; Koby et al., 2010; Ozcetin et al., 2009; Portuguese et al., 2007; Reilly et al., 1999; Salmon et al., 2003; Snyder et al., 1994). Across the 10 studies citing rates of physical abuse in childhood, the average rate was 54%. Regarding physical abuse/assault in adulthood, rates of 13-52% have been reported in the literature (Bowman, 1993; Bowman & Markand, 1996; 1999; Koby et al., 2010; Reilly et al., 1999; Salmon et al., 2003), with an average rate of 30%. Bowman and Markand (1996) and Thomas et al. (2013) found higher rates of physical abuse in female patients. However, Koby and colleagues (2010) reported that physical abuse was more frequently claimed by male patients.

Significantly higher rates of physical abuse have been reported in DS samples compared to control groups, including those with ES (Akyuz et al., 2004; Alper et al., 1993; Kaplan et al., 2013; Koby et al., 2010; Salmon et al., 2003; Strutt et al., 2011a,b; Tojek et al., 2000) and healthy controls (Bakvis, Roelofs, et al., 2009; Ozcetin et al., 2009); although other studies have failed to find such differences (Arnold & Privitera, 1996; Bakvis, Spinhoven, Giltay, et al., 2010; Berkhoff et al., 1998; Kuyk et al., 1999;

Litwin & Cardeña, 2000; Proença et al., 2011).

### ***Psychological abuse***

Rates of lifetime psychological/emotional abuse are high in patients with DS, ranging from 37-74% of patients reporting such experiences, with an average of 49% (Bakvis, Roelofs, et al., 2009; Bakvis, Spinhoven, Giltay, et al., 2010; Driver-Dunckley et al., 2011; Kuyk et al. 1999; Strutt et al., 2011a,b). Between 33 and 61% of patients with DS report childhood psychological abuse or neglect (Akyuz et al., 2004; Bowman & Markand, 1999; Portuguese et al., 2007; Reilly et al., 1999; Salmon et al., 2003). Furthermore, reports of adulthood psychological abuse indicated a rate of around 38% (Reilly et al., 1999; Salmon et al., 2003). Some investigators have also found that rates of emotional abuse and/or neglect are higher in patients with DS than patients with ES (Proença et al., 2011; Salmon et al., 2003; Strutt et al., 2011a,b), conversion movement disorders (Driver-Dunckley et al., 2011), and healthy controls (Ozcetin et al., 2009), although not in every study (Kuyk et al., 1999).

### ***Post-traumatic stress disorder (PTSD)***

The review by Fiszman et al. (2004) noted that rates of both lifetime and current PTSD were higher in DS patients than in the general population. Lifetime or current PTSD has been reported in between 0.5% and 76.9% of patients with DS (Abubakr et al., 2003; Arnold & Privitera, 1996; Bowman, 1993; Bowman & Markand, 1996; Chen & Izadyar, 2009; Dikel et al., 2003; Elliott & Charyton, 2014; Hingray et al., 2011; Myers et al., 2013; O'Brien et al., 2015; Rosenberg et al., 2000; Scévola et al., 2013). PTSD has been found to be more common in patients with DS relative to those with ES in some studies (Elliott & Charyton, 2014; Koby et al., 2010; Salinsky et al., 2012), although this difference was only observed in male patients in the study of Koby et al. (2010). Moreover, the sample studied by Salinsky et al. (2012) consisted of war veterans only, which limits the generalisability of those findings. Nevertheless, Rosenberg et al. (2000) reported that the factor with most predictive power for a diagnosis of DS (rather than ES) was PTSD diagnosis. Similarly, PTSD was the only psychiatric diagnosis that significantly distinguished veterans with DS from those with ES in a regression analysis described by Salinsky et al. (2012).

### ***Evaluation of research on trauma and DS***

There is considerable variability in the quality of research in this area. One important limitation of many studies is the omission of a comparison group, particularly as population base-rates have not been provided. There is also a wide range of techniques used to assess trauma history, frequently involving retrospective review of case notes and self-report measures, both of which have limitations. Only one study (Betts and Boden, 1992b) included the criterion of independent corroboration (at the time of disclosure) for reports of abuse. Furthermore, only a few studies have blinded the investigator(s) to diagnosis or the hypotheses of the study (Arnold and Privitera, 1996; Rosenberg et al., 2000). Another issue is that some investigators do not report separate statistics for different types of abuse which can make interpretation difficult (e.g. Elliott and Charyton, 2014; Krahn et al., 1997; Wolf et al., 2015). Traumatic and stressful life events are also confounded in some studies (e.g. Gambini et al., 2014).

Some investigators failed to match comparison groups on gender (e.g. Alper et al., 1993; Litwin & Cardeña, 2000). Furthermore, several studies included small sample sizes of less than 20 patients, thereby limiting statistical power (Arnold & Privitera, 1996; Berkhoff et al., 1998; Litwin & Cardeña, 2000; Rosenberg et al., 2000). In contrast to many others, the studies carried out in Iran (Asadi-Pooya, Emami, & Emami, 2014), China (An et al., 2010) and Spain (Baillés et al., 2004) reported much lower rates of sexual and physical abuse in their samples of patients with DS. Rates of occurrence/disclosure of abuse may have been affected by cultural factors in these samples.

Sharpe and Faye (2006) noted that many of the samples included in past studies have been recruited in specialist medical settings, which might have biased the samples towards particularly severe, chronic or complex cases. Van Merode et al. (2004) circumvented this problem by conducting a controlled prospective study of individuals experiencing their first seizure from the general population. Further studies of this nature would be valuable. Nonetheless, the research literature suggests that traumatic experiences are an important contributing factor in DS. Traumatic experiences, particularly in childhood, have been proposed to be important predisposing/causal factors in several aetiological models of DS (Bodde et al., 2009; Mellers, 2009; Oto &

Reuber, 2014; Reuber, 2009). The current review supports this hypothesis.

### **2.2.2. Stressful life events**

Patients with DS report a variety of stressful life events. For example, stressful circumstances were identified in 58% of one sample of patients with DS (Alessi and Valente, 2013). Furthermore, Bodde et al. (2013) reported elevations in scores on the Everyday Problem Checklist in their DS group, relative to normative standards. Stressors described in the literature include divorce, bereavement, major surgery/illness, accidents (e.g. road traffic), legal issues, encountering a former abuser, general relationship problems, work-related problems, inescapable negative life circumstances, physical assaults/injuries and some 'positive' events such as changes in job status/employment, and expecting a baby (Alessi & Valente, 2013; Bowman, 1993; Bowman & Markand, 1996; 1999; Dickinson et al., 2011; Grimaldi et al., 2009).

Patients with DS perceive significantly more stress in daily life (Frances et al., 1999), and report more psychological distress in response to adverse life events, relative to healthy controls (Testa et al., 2012). Moreover, relative to those with ES, patients with DS receive higher scores on the Stress subscale of the Personality Assessment Inventory (Testa et al., 2011), endorse a greater number of stressful life events, perceive them as more stressful (Tojek et al., 2000), and report higher levels of worry and tension (Krishnamoorthy et al., 2001).

More life events in the year prior to disorder onset are reported by patients with DS than control groups with ES (Binzer et al., 2004) or medically unexplained motor disorders (Stone, Sharpe, & Binzer, 2004). Driver-Dunckley et al. (2011) found that 63% of their sample of DS patients could identify at least one stressor that was perceived as triggering or worsening their seizures. Approximately 90% of a Turkish sample of DS patients were able to identify stressful events or stimuli prior to losing consciousness (Almis et al., 2013). Therefore, in at least some patients with DS, acute or chronic stressors may contribute to seizure initiation on an ongoing basis. However, not all patients with DS identify stress or emotion as seizure triggers, and they have been found to be more likely to deny stressful life circumstances than patients with ES (Stone, Binzer, & Sharpe, 2004).

Stressful or adverse life events are often discussed as precipitating factors in previous accounts of DS (Mellers, 2009; Oto & Reuber, 2014; Reuber, 2009). However, multiple or chronic stressors occurring during development could also predispose to the development of DS, perhaps by influencing stress reactivity, psychopathology or dissociative/somatoform tendencies. Moreover, psychological perspectives on stress assume an important role of cognitive factors such as appraisal and self-efficacy (i.e. Lazarus & Folkman, 1987). There is some literature suggesting that coping strategies and illness-beliefs are less adaptive in patients with DS than comparison groups (see section 2.3.1. and 2.3.5). Therefore, it seems that life events/circumstances may interact with cognitive factors and maladaptive coping skills in elevating patients' stress levels and contributing towards DS.

### **2.2.3. Relationship dysfunction**

Signs of dysfunction have been reported within the families of patients with DS (Hovorka et al., 2007; Lancman et al., 1993; Moore & Baker, 1997; Silva et al., 2001). The characteristics differing between the families of patients with DS and control groups include: higher rates of divorce/separation (Stone, Sharpe, & Binzer, 2004), emotional over-involvement (Salmon et al., 2003; Stanhope et al., 2003), less cohesion (P.M. Moore et al., 1994), less emphasis on moral/religious values (P.M. Moore et al., 1994), reduced value on intellectual pursuits (P.M. Moore et al., 1994), lack of emotional warmth (Binzer et al., 2004; Stone, Sharpe, & Binzer, 2004), abnormal role definition and boundaries (Krawetz et al., 2001), atypical expressiveness/communication (Salmon et al., 2003; Stanhope et al., 2003), rejection (Binzer et al., 2004; Stone, Sharpe, & Binzer, 2004), overall family dysfunction (LaFrance et al., 2011), somatisation in family members (Wood et al., 1998), criticism (Wood et al., 1998), high levels of control (Salmon et al., 2003), and general family distress (Wood et al., 1998).

Tojek and colleagues (2000) reported that patients with DS described significantly poorer childhood relationships with their fathers than controls with ES. Moreover, 'unspeakable dilemmas' and emotional 'double-binds' have been observed by some investigators (Bowman & Markand, 1999; J.L. Griffith et al., 1998). Patients with DS



may also exhibit dysfunctional (fearful) attachment styles relative to patients with ES (Holman et al., 2008), although another study (Lally et al., 2010) failed to replicate this finding.

It is important to note that some of the above-mentioned studies found differences in patient-reported perceptions of family functioning (e.g. Binzer et al., 2004; Salmon et al., 2003), whereas others examined the perceptions of family members themselves (e.g. Krawetz et al., 2001; Wood et al., 1998). It is possible that both types of rating are somewhat biased by the respondent, and so more observational studies with objectively defined criteria and impartial raters might be beneficial (e.g. Stanhope et al., 2003). Nevertheless, family dysfunction, particularly in childhood may serve as an important predispositional factor (Mellers, 2009), and/or mediate the influence of abuse (Salmon et al., 2003).

Problems in adult interpersonal relationships are also evident in some patients with DS. For example, 59% of one adult sample claimed to have at least one relationship currently causing emotional conflict (Silva et al., 2001). Schramke et al. (2010) noted that 'marital instability' significantly predicted a diagnosis of DS. Relationship disturbances in adulthood (e.g. workplace bullying, marital conflict) could potentially act as the precipitating stressor in disorder onset, and/or may trigger DS on an ongoing basis. Moreover, Reuber (2009) discusses social isolation and dependence on others as potential consequences of living with DS, which may also come to perpetuate the disorder.

## **2.3. Psychological factors**

### **2.3.1. Coping styles**

Patients with DS report more use of escape/avoidant coping strategies, relative to healthy controls (Cronje & Pretorius, 2013; Frances et al., 1999; Goldstein et al., 2000; Myers, Fleming, et al., 2013). Bodde and colleagues (2013) found that avoidant behavioural strategies were preferred by their sample of patients with DS. Moreover, Goldstein and Mellers (2006) noted more agoraphobic-type avoidant behaviours in

their sample of patients with DS, relative to those with ES. However, some studies have failed to find differences between patients with DS and ES on coping measures (Frances et al., 1999; O'Brien et al., 2015; van Merode et al., 2004).

'Distancing' strategies are also reported more commonly in DS patients, compared to healthy controls (Cronje & Pretorius, 2013); but not relative to patients with ES (Frances et al, 1999). Dimaro et al. (2014), more recently, observed elevated levels of 'experiential' avoidance in patients with DS, relative to those with ES and healthy control participants. This type of avoidance includes cognitive avoidance of unwanted or unpleasant psychological states, such as negative emotions and mental distress (similarly to distancing).

Interestingly, Goldstein et al. (2000) observed a positive correlation between escape/avoidance strategies and trait dissociation scores in their DS group, suggesting that avoidant responses to stressful situations were linked to a tendency to dissociate. Moreover, patients with DS are also less likely to use active or 'planful problem-solving' strategies than healthy individuals (Frances et al., 1999; Goldstein et al., 2000; Testa et al., 2012). Other authors have reported reduced use of 'task-oriented' coping techniques (Myers, Fleming, et al., 2013).

Individuals with DS, therefore, seem more likely to respond to distressing situations/experiences with behavioural, cognitive and affective avoidance. The use of avoidant coping strategies has been conceptualised as a perpetuating factor by some authors (e.g. Mellers, 2009; Reuber, 2009). Avoidant/escape coping strategies may contribute to the maintenance of current anxiety levels (Frances et al., 1999), and thus might contribute to ongoing seizure reoccurrence. However, a pre-existing tendency towards avoidance of undesired emotions/situations also might contribute to the risk for developing DS, perhaps by increasing a tendency towards somatoform and dissociative symptoms more generally.

### **2.3.2. Psychopathology**

#### ***Psychiatric diagnoses / history***

Patients with DS often present with one or more current/past psychiatric diagnoses

(Abubakr et al., 2003; Ahmedani et al., 2013; Baillés et al., 2004; Baslet, Roiko, & Prensky, 2010; Bora et al., 2011; Bowman, 1993; 2001; d'Alessio et al., 2006; Direk et al., 2012; Galimberti et al., 2003; Gambini et al., 2014; Gazzola et al., 2012; Hixson et al., 2006; M.D. Holmes, Dodrill, et al., 2001; Jones et al., 2010; Kanner et al., 1999; Krishnamoorthy et al., 2001; Kristensen & Alving, 1992; Marchetti et al., 2009; Mazza et al., 2009; Mitchell et al., 2012; Mökleby et al., 2002; Moore & Baker, 1997; O'Sullivan et al., 2007; Prigatano et al., 2002; Schramke et al., 2010; Seneviratne et al., 2011; Snyder et al., 1994; Turner et al., 2011).

Furthermore, comorbid formal psychiatric diagnoses have been reported to be elevated in patients with DS, relative to those with somatisation disorders (Mökleby et al., 2002) and ES (e.g. Galimberti et al., 2003; Salinsky et al., 2012; Scévola et al., 2013; Szaflarski et al., 2003), although not consistently (Arnold & Privitera, 1996; Binzer et al., 2004; Direk et al., 2012). Additional psychiatric diagnoses are associated with poorer outcomes in patients with DS (see Chapter 1, section 1.6).

However, a variety of techniques have been used to assess psychiatric diagnoses across studies, including retrospective examination of clinical notes, self-report questionnaires and structured or unstructured clinical interviews. Furthermore, current and historical psychiatric diagnoses are not always differentiated. Intellectual disability is reported as a psychiatric diagnosis in some studies, but not others, and crucially, studies also differ with regard to whether the diagnosis of DS itself is included in the rates of psychiatric morbidity reported. Many investigators also only assess a limited number of psychiatric diagnoses, usually those known to be common in DS.

Additional psychiatric disorders in DS have been argued to be causal of the seizure disorder by some authors (e.g. Marchetti et al., 2009). However, an alternate viewpoint is that the additional disorders are associated with, but not causal of DS. For example, depressive symptoms might develop or worsen as a result of living with DS, rather than DS being caused by pre-existing depression. Depression may share some causal factors with DS and once developed, could trigger or exacerbate the occurrence of DS by increasing the individual's general emotional distress. Furthermore, it should be noted that many patients with DS have no other psychiatric

diagnoses aside from the diagnosis of DS itself (e.g. O'Brien et al., 2015).

### ***Psychopathological symptoms***

Inventories such as the Symptom Check List – Revised (SCL-90-R; Derogatis, 1994) or the Brief Symptom Inventory (BSI; Derogatis, 1993) have been used to examine symptoms of general psychopathology in DS samples. Such measures examine a range of psychopathological symptoms (e.g. somatisation, depression, anxiety). Patients with DS tend to receive higher scores than control groups on such measures, suggesting elevated psychopathology in one or more domains (Kuyk et al., 1999; Lally et al., 2010; Prueter et al., 2002; Reuber, House, et al., 2003; van Merode et al., 2004).

The Personality Assessment Inventory (PAI; Morey, 1991), Minnesota Multiphasic Personality Inventory (MMPI) or MMPI-2 (Hathaway & McKinley, 1943; 1989), and the Millon Multiaxial Inventory of Assessments (MCMI; Millon, 1977) have also been administered by some investigators. Scales on the MMPI/MMPI-2 are often elevated in patients with DS (e.g. Baillés et al., 2004; M.C. Brown et al., 1991). The most commonly elevated scales are Hypochondriasis and Hysteria, with scores often higher in patients with DS relative to those with ES (Binder et al., 2000; Bodde et al., 2011; Derry & McLachlan, 1996; Drake et al., 1993; Hixson et al., 2006; Kalogjera-Sackellares & Sackellares, 1997; Owczarek, 2003; Owczarek and Jędrzejczak, 2001; Slater et al., 1995; Strutt et al., 2011b; Wilkus & Dodrill, 1989). On the PAI, one or more of the subscales (Conversion, Somatisation, Health Concerns) on the Somatic Concerns scale are often found to be elevated relative to norms or control groups (e.g. Gale & Hill, 2011; 2012; O'Brien et al., 2015; Testa & Brandt, 2010; Wolf et al., 2015). Moreover, scores on such measures often conform to specific patterns in patients with DS, such as the 'conversion V' on the MMPI (e.g. Drake et al., 1992; Slater et al., 1995) and the 'NES indicator' pattern on the PAI (M.T. Wagner et al., 2005).

An important issue in interpreting these findings is that items on some scales of the MMPI or PAI may relate to the seizure symptoms themselves and so might confound results. In addition, interpretation of MMPI and PAI scores can be problematic because the measures examine both personality characteristics and symptoms of psychopathology. Reuber, Pukrop, et al. (2004) further discuss some of the

shortcomings of using the MMPI in this population.

### ***Affective symptoms and disorders***

Patients with DS often report depressive symptomology (Ettinger, Devinsky, et al., 1999; Farnam et al., 2010; Hixson et al., 2006; Mazza et al., 2009; Mökleby et al., 2002; P.M. Moore et al., 1994; Prueter et al., 2002; Strutt et al., 2011a,b; Szaflarski & Szaflarski, 2004; Testa et al., 2011; 2012; A.W. Thompson et al., 2010; M.T. Wagner et al., 2005; Xue et al., 2013). Scores on depression inventories have been found to be elevated compared to healthy controls (Bewley et al., 2005; Hixson et al., 2006; Mazza et al., 2009; Mökleby et al., 2009; P.M. Moore et al., 1994; O'Brien et al., 2015; Testa et al., 2011; 2012; Urbanek et al., 2014; Xue et al., 2013) and neurology patients (Farnam et al., 2008). Furthermore, higher depression scores predict reduced quality of life in patients with DS (Karakis et al., 2014).

Several studies have indicated that patients with DS report more total or somatic depressive symptoms than patients with ES (Asmussen et al., 2009; Karakis et al., 2014; LaFrance et al., 2011; Mazza et al., 2009; Szaflarski & Szaflarski, 2004; Testa et al., 2011; A.W. Thompson et al., 2010; M.T. Wagner et al., 2005). However, a comparable number of studies have found no such differences (Bewley et al., 2005; Hixson et al., 2006; P.M. Moore et al., 1994; Prigatano & Kirlin, 2009; Salmon et al., 2003; Strutt et al., 2011b; Testa et al., 2012; Wolf et al., 2015). Patients with DS also report comparable levels of depression as patients with somatoform disorders (Mökleby et al., 2002) and conversion movement disorders (Grimaldi et al., 2009; Hopp et al., 2012). However, patients with ES (Hoppe & Elger, 2011; Kanner et al., 2012), somatoform (e.g. De Waal et al., 2004), and conversion movement disorders (e.g. Feinstein et al., 2001) may also report elevated rates of depression relative to healthy individuals.

Patients with DS often meet criteria for a formal diagnosis of depression (Abubakr et al., 2003; Alessi & Valente, 2013; Arnold & Privitera, 1996; Bowman 1993; Bowman & Markand, 1996; Conwill et al., 2014; Driver-Dunckley et al., 2011; Elliott & Charyton, 2014; Ettinger, Dhoon, et al., 1999; Hixson et al., 2006; Jones et al., 2010; Kanner et al., 1999; 2012; Lempert & Schmidt, 1990; Marchetti et al., 2009; Mökleby et al., 2002; O'Sullivan et al., 2007; Patidar et al., 2013; Schramke et al., 2010; Snyder et al., 1994;

Strutt et al., 2011a,b). Fiszman and Kanner (2010) reviewed findings from studies using DSM diagnostic criteria, and reported prevalence rates for depression in patients with DS ranging from 21-60%. Diagnoses of depression have been found at higher rates in patients with DS than ES in some studies (e.g. Elliott & Charyton, 2014; Schramke et al., 2010), but not all (e.g. Scévola et al., 2013).

Bipolar disorder is generally less common in DS than depression (Alessi & Valente, 2013; Bowman, 1993; Bowman & Markand, 1996), but it has been reported significantly more commonly in some samples of patients with DS compared to those with ES (Elliott & Charyton, 2014; Salinsky et al., 2012). Suicide attempts, suicidal ideation and self-harm behaviors have been reported a number of times in DS samples (Akyuz et al., 2004; Alessi & Valente, 2013; d'Alessio et al., 2006; Duncan et al., 2011; Elliott & Charyton, 2014; Ettinger, Devinsky, et al., 1999; Moore & Baker, 1997; Rechlin et al., 1997; Snyder et al., 1994; Stewart et al., 1982; Testa et al., 2011; A.W. Thompson et al., 2010).

Mood disturbance may be a trait characteristic that predisposes an individual to developing DS. Furthermore, an episode of major depression, for example, might precipitate the onset of DS. Ongoing affective disturbance may also perpetuate the disorder, contributing to the emotional distress that might trigger individual seizures. Finally, affective symptoms such as depression may well be a consequence of living with DS for some patients.

### ***Anxiety symptoms and disorders***

Symptoms of anxiety have been found to be elevated in patients with DS relative to healthy participants (Bewley et al., 2005; Hixson et al., 2006; Mökleby et al., 2002; O'Brien et al., 2015; Testa et al., 2011; 2012; Urbanek et al., 2014; Xue et al., 2013). However, when patients with ES and DS have been compared on self-reported anxiety, findings have been mixed. A number of investigators have reported comparable scores (Bewley et al., 2005; Dimaro et al., 2014; Hixson et al., 2006; Prigatano & Kirlin, 2009; Salmon et al., 2003; Strutt et al., 2011a; Wolf et al., 2015), whereas others have reported higher anxiety scores in DS samples relative to ES (Holman et al., 2008; Karakis et al., 2014; Lawton, Baker, & Brown, 2008; P.M. Thompson, Batzel, & Wilkus,

1992; A.W. Thompson et al., 2010; van Merode et al., 2004). Conversely, Akyuz et al. (2004) found the opposite pattern (higher scores in the ES group).

It has been suggested that patients with DS experience increased physiological anxiety symptoms generally (Testa et al., 2011; A.W. Thompson et al. 2010) and during their attacks than ES patients, despite not necessarily experiencing heightened subjective fear (Frolov, Korinevskaya, & Vorob'eva, 2003; Goldstein & Mellers, 2006). Patients with DS report more peri-ictal panic symptoms than those with ES (Hendrickson et al., 2014). Furthermore, patients with DS also score higher on a measure of 'fear sensitivity' (Hixson et al., 2006).

Current or previous diagnoses of anxiety disorders have been observed frequently in patients with DS (Alessi & Valente, 2013; Bowman & Markand, 1996; Conwill et al., 2014; Dworetzky et al., 2005; Galimberti et al., 2003; Hixson et al., 2006; Hovorka et al., 2007; Kuyk et al., 2003; LaFrance & Syc, 2009; LaFrance et al., 2011; Patidar et al., 2013; Schramke et al., 2010; P.M. Thompson, et al., 1992; A.W. Thompson et al., 2010), although the prevalence of anxiety disorders is more variable across studies than depression (Fisman & Kanner, 2010). Nonetheless, some studies have noted higher rates of current or previous anxiety disorders in DS patients relative to patients with ES (Elliott & Charyton, 2014; Krishnamoorthy et al., 2001; LaFrance et al., 2011; Scévola et al., 2013; Schramke et al., 2010). Anxiety disorder has also been reported to significantly predict a diagnosis of DS rather than ES (Elliott & Charyton, 2014). Panic disorder (Arnold & Privitera, 1996; Krishnamoorthy et al., 2001; Schramke et al., 2010; Snyder et al., 1994) and PTSD (Arnold & Privitera, 1996; Bowman, 1993; Bowman & Markand, 1996; d'Alessio et al., 2006; Dikel et al., 2003; Dworetzky et al., 2005; Fisman & Kanner, 2010; Rosenberg et al., 2000; Scévola et al., 2013) are commonly comorbid with DS.

As with depression, elevated levels of anxiety may act as a predisposing factor in DS. Furthermore, high levels of anxiety could act as a precipitant to the onset of the disorder and may also trigger individual episodes. Anxiety may also serve to perpetuate the disorder, by increasing the likelihood of using psychological and behavioural avoidance strategies, and by contributing to raised levels of autonomic

arousal.

### ***Medically unexplained symptoms and disorders***

Additional MUS are often reported by patients with DS (Alessi & Valente, 2013; Bowman, 1993; Dimaro et al., 2014; Dixit et al., 2013; Duncan et al., 2011; Salmon et al., 2003; P.M. Thompson, et al., 1992; A.W. Thompson et al., 2010). For example, Duncan et al. (2011) reported that 57.4% of their sample of patients with DS experienced additional functional symptoms. Patients with DS report more somatoform symptoms than patients with ES (R.J. Brown et al., 2013; Dimaro et al., 2014; Dixit et al., 2013; Hill & Gale, 2011b; Reuber, House, et al., 2003; Testa et al., 2011; A.W. Thompson et al., 2010) and healthy controls (Dimaro et al., 2014; Testa et al., 2011). Scores on measures of somatisation predict a diagnosis of DS rather than ES (Dimaro et al., 2014; Hill & Gale, 2011b; Reuber, House, et al., 2003). Importantly, higher somatisation scores have been linked to worse outcomes (Reuber, House, et al., 2003) and reduced quality of life (LaFrance & Syc, 2009; Wolf et al., 2015) in patients with DS. In addition, somatisation has been reported to mediate the relationship between the diagnosis of DS and reduced quality of life (Wolf et al., 2015).

Patients with DS often present with additional conversion or somatoform diagnoses, or meet criteria for these (Alessi & Valente, 2013; Bowman & Markand, 1996; d'Alessio et al., 2006; Dixit et al., 2013; Galimberti et al., 2003; Krishnamoorthy et al., 2001; LaFrance & Syc, 2009; Mökleby et al., 2002; O'Sullivan et al., 2007). Somatoform disorders have been found at higher rates in patients with DS than those with ES (Dixit et al., 2013), or those with mixed ES and DS (Kuyk et al., 2003). Elevated levels of bodily awareness were also observed in one group of patients with DS, relative to patients with ES (Tojek et al., 2000). Medically unexplained pain is a particularly common comorbid diagnosis in patients with DS (Bowman & Markand, 1996; Driver-Dunckley et al., 2011; Elliott & Charyton, 2014; Mökleby et al., 2002; O'Sullivan et al., 2007). Several authors have reported higher rates of chronic pain (Drane et al., 2006; Dworetzky et al., 2005; Elliott & Charyton, 2014; Gazzola et al., 2012) and fibromyalgia (Drane et al., 2006) in patients with DS relative to those with ES, and the presence of chronic pain has also been found to predict a diagnosis of DS rather than ES (Elliott & Charyton, 2014).



A general tendency to experience MUS may act as a predisposing factor for the development of DS. However, an episode of severe/multiple MUS might precipitate the initial occurrence of DS. Furthermore, after the onset of the seizure disorder, additional MUS may perpetuate DS by contributing to high levels of emotional distress and/or disproportionately focusing attention on somatic processes. Bodde et al. (2009) also note that dissociation and somatisation may act as the triggering mechanism for DS, whereby emotional/mental states are transformed into a seizure (see Chapter 3).

### ***Intellectual disability***

Intellectual disability (ID) is identified in a proportion of patients with DS. For example, rates of approximately 9-30% (Duncan & Oto, 2008; Elliott & Charyton, 2014; Gambini et al., 2014; Moore & Baker, 1997) have been reported in previous samples. Furthermore, Duncan and Oto (2008) assessed potential differences between DS patients with and without ID. It was reported that comorbid ES, AED use at diagnosis, history of 'pseudostatus', and immediate situational triggers for individual DS were more common in the ID group relative to the non-ID group. Pre-existing cognitive impairment could, therefore, act as a predisposing factor for developing DS (Bodde et al., 2009; Reuber, 2009), and may represent a unique subgroup of patients, with different risk factors and triggering mechanisms than patients without ID (Duncan & Oto, 2008).

### **2.3.3. Personality traits and disorders**

Studies report rates of personality disorder in the range of approximately 18-85% in patients with DS (Arnold & Privitera, 1996; Baillés et al., 2004; Bowman & Markand, 1996; d'Alessio et al., 2006; Direk et al., 2012; Elliott & Charyton, 2014; Galimberti et al., 2003; Harden et al., 2009; Hovorka et al., 2007; Kanner et al., 1999; Kuyk et al., 2003; O'Brien et al., 2015; Rechlin et al., 1997; Salinsky et al., 2012; Scévola et al., 2013). Rates of personality disorder have been found to be higher in DS patients than control groups (Binzer et al., 2004; Direk et al., 2012; Elliott & Charyton, 2014; Salinsky et al., 2012), although not consistently (i.e. Arnold and Privitera, 1996; Scévola et al., 2013). Nonetheless, individuals with comorbid DS and personality disorder seem to have

poorer outcomes than those without personality disorder (Drake et al., 1992).

‘Cluster B’ personality disorders are the most commonly reported, particularly borderline (BPD) and histrionic personality disorders (Arnold & Privitera, 1996; Baillés et al., 2004; Binzer et al., 2004; Bowman & Markand, 1996; d’Alessio et al., 2006; Direk et al., 2012; Galimberti et al., 2003; Harden et al., 2009; Hovorka et al., 2007; Lacey, Cook, & Salzberg, 2007; LaFrance & Syc, 2009; O’Brien et al., 2015; Rechlin et al., 1997; Scévola et al., 2013; Stone, Sharpe, & Binzer, 2004; P.M. Thompson, et al., 1992; Turner et al., 2011). Higher rates of BPD have been observed in patients with DS relative to those with motor conversion symptoms (Stone, Sharpe, & Binzer, 2004) and patients with ES (Binzer et al., 2004; Scévola et al., 2013).

Patients with DS share a number of clinical similarities with patients with BPD, such as trauma history, high levels of dissociative symptoms, affect dysregulation and increased risk of suicidal thoughts and behaviours (Lacey et al., 2007). Nevertheless, several authors have also reported ‘cluster C’ personality disorders in patients with DS, most often of the avoidant type (d’Alessio et al., 2006; Arnold & Privitera, 1996; Bowman & Markand, 1996; Direk et al., 2012; Hovorka et al., 2007; Kanner et al., 1999), although rates of ‘cluster C’ personality disorders have been found to be higher in patients with mixed ES and DS compared to those with DS only (Kuyk et al., 2003).

Some investigators have explored personality traits in patients with DS using dimensional self-report measures. For example, Reuber, Pukrop, et al. (2004) found a greater extent of personality abnormality in patients with DS relative to patients with ES, as assessed with the Dimensional Assessment of Personality Pathology (DAPP; Livesley & Jackson, 2002). Borderline personality features were the most common pattern in the DS group. Furthermore, one study reported differences between patients with ES and DS on the ‘big five’ personality dimensions (i.e. neuroticism, openness, agreeableness, conscientiousness, extraversion), measured with the NEO-PI-R (Cragar et al., 2005). However, no differences on this measure were reported in another study (Testa & Brandt, 2010). More studies of normal personality variation in this group might be warranted.

As noted in section 2.3.2, responses on measures such as the MMPI/MMPI-2 and the PAI have been examined in patients with DS in numerous studies. The findings usually include atypical scores on several dimensions in DS samples, mostly linked to somatisation, conversion and concerns about bodily health. However, as previously mentioned, it is difficult to determine the extent to which these scales reflect current psychopathology or more stable personality traits; therefore, alternative and more focused personality measures are preferable.

Particular personality characteristics (e.g. emotional dysregulation, somatisation, avoidance) may serve as predisposing factors in some patients who develop DS. However, it could also be argued that personality features might perpetuate the disorder. For example, individuals high in trait emotional dysregulation may be more likely to encounter stressful situations (e.g. interpersonal conflict) and respond with higher levels of distress, thereby creating more situational/emotional triggers for the reoccurrence of seizures. On the other hand, avoidant tendencies are likely to negatively affect general functioning (e.g. reducing social, occupational or educational functioning), which could maintain elevated levels of emotional distress, and thus perpetuate the disorder. Nonetheless, it seems that no single personality pathology uniquely characterises patients with DS.

#### **2.3.4. Exposure to symptom ‘models’**

It is not unusual for patients with DS to report the presence of epilepsy in their family members (Asadi-Pooya & Emami, 2013; Dickinson et al., 2011). Rates of reported ES in family members of patients with DS range from around 15 to 50% (Asadi-Pooya, Emami, & Emami, 2014; Bodde et al., 2007; LaFrance & Syc, 2009; LaFrance et al., 2011; Lancman et al., 1993; Reuber et al., 2011; Moore and Baker, 1997; Wadwekar et al., 2014), although most are between 30-50%. Patients with DS are more likely to report having a relative with epilepsy than patients diagnosed with ES (Gazzola et al., 2012; Locke et al., 2006; Schramke et al., 2010; Tojek et al., 2000) or general psychiatric outpatients (Jawad et al., 1995), although not in every study (i.e. Bautista, Gonzales-Salazar, & Ochoa, 2008).

It is possible that in some cases, the family member may in fact also be displaying DS

rather than ES. Nevertheless, the occurrence of seizures in relatives may contribute to the development of DS in patients exposed to these, by serving as a shaping (predispositional) factor (Bodde et al., 2009; Mellers, 2009; Reuber, 2009). Exposure to such symptom models may influence the development of the specific form of symptoms with which patients present.

### **2.3.5. Cognitive factors: locus of control, self-efficacy and illness-representations**

Locus of control (LOC) refers to the extent to which an individual attributes events to internal or external causes (Rotter, 1990). Patients with DS report a more externally-oriented health-related LOC than healthy controls (Goldstein et al., 2000), and a more external general (i.e. non-health specific) LOC compared to patients with ES (Stone, Binzer, & Sharpe, 2004). These findings suggest that patients with DS are more likely to perceive external causes for events, with reduced perception of internal control or influence. In contrast, P.M. Moore and colleagues (1994) found no significant differences between patients with DS, ES or healthy controls on a measure of LOC. It is possible that the different measures used between studies might account for the variable findings.

Nonetheless, other differences have emerged in the beliefs and representations that patients with DS have about their disorder and/or illness in general. Patients with DS ascribe greater influence of other people on their seizure disorder (Strutt et al., 2011b), are less likely to attribute their illness to psychological factors, and show more denial of stressful life events (Stone, Binzer, & Sharpe, 2004), relative to patients with ES. However, some authors have reported broadly similar illness representations in patients with DS and control groups. For example, Hopp and colleagues (2012) found that patients with DS had similar levels of (health-related) self-efficacy to patients with movement disorders in a number of domains (managing disease and symptoms, doing chores, exercise and social activities).

Furthermore, an experimental study of implicit attitudes to illness failed to identify any specific biases in the DS group relative to those with ES or healthy control participants (Testa & Brandt, 2010). Whitehead and colleagues (2013) also identified

few differences in illness beliefs between patients with DS and ES. Nevertheless, important differences between patients with DS and their neurologists emerged in the latter study. For example, neurologists were more likely to attribute DS to psychological factors, whereas patients were more likely to perceive an influence of physical factors on the disorder.

In summary, patients with DS generally have a more externally-oriented (general and health-related) LOC than control groups, and are less likely to perceive psychological influences on their seizures than patients with ES and expert clinicians. Such beliefs and tendencies may act as perpetuating factors in DS (Mellers, 2009; Reuber, 2009).

### **2.3.6. Neuropsychological / cognitive abilities**

There have been some reports of reduced neurocognitive functioning in DS samples relative to normative standards (Kalogjera-Sackellares & Sackellares, 1999; Reuber, Fernández, Helmstaedter, Qurishi, & Elger, 2002; Snyder et al., 1994; Strutt et al., 2011a) or non-clinical control groups (Almis et al., 2013; Binder et al., 1998; Bodde et al., 2013; O'Brien et al., 2015; van der Kruijs et al., 2012). Differences in various cognitive domains have been reported, including aspects of memory, general intellectual functioning, executive skills, motor performance, general processing speed, attention, and verbal fluency. However, performance in DS samples is often in the low average or borderline ranges, rather than significantly impaired as in ID.

Patients with DS perform equivalently or worse than patients with ES on some neuropsychological tests (Binder et al., 1998; Bortz et al., 1995; M.C. Brown et al., 1991; Dodrill, 2008; Drane et al., 2006; Holman et al., 2008; K.A. McNally et al., 2009; Slater et al., 1995; van Merode et al., 2004; Wilkus, Dodrill, & Thompson, 1984; Wilkus & Dodrill, 1989), including tests of general intellectual functioning, verbal, visual and autobiographical memory, attention, and set-shifting. However, it is possible that the deficits observed in patients with DS may be associated with reduced effort, psychopathological factors, seizure occurrence, or decision-making deficits, rather than due to organic brain dysfunction as in ES (Binder & Campbell, 2004; Binder et al., 1998; Drane et al., 2006; K.A. McNally et al., 2009; Myers et al., 2014; Prigatano & Kirlin, 2009; Williamson et al., 2005; 2012), although the findings on reduced effort are

inconsistent (Cragar et al., 2006; Dodrill, 2008).

Performance on neuropsychological tests has been found to be associated with clinical characteristics in patients with DS, including seizure types (worse performance in patients with major motor seizures; Hill & Gale, 2011a), duration of DS disorder, age at seizure onset, and seizure frequency (L.C. Black et al., 2010). Moreover, inaccuracies in self-reported cognitive functioning have been reported in this group, often involving underestimation of their abilities (Breier et al., 1998; Fargo et al., 2004).

The mild cognitive deficits observed in patients with DS may be related to neurological factors (i.e. minor head injury). The deficits could also be a consequence of medication use (i.e. AEDs), psychopathology (i.e. depression), or other psychological factors (i.e. motivation), alone or in combination. However, the possibility remains that pre-existing subtle deficits in cognitive functioning might predispose toward developing DS in some individuals. Moreover, such deficits might also precipitate or perpetuate the disorder by negatively affecting information processing and/or responding in complex or challenging situations.

#### **2.4. Summary and evaluation of literature review**

A range of variables contribute to the occurrence of DS and no one universal pattern of biopsychosocial factors is consistently associated with the disorder. In terms of predisposing factors, the following are likely candidates: a propensity for somatisation/dissociation, psychopathology, personality characteristics, developmental adverse life experiences and/or family dysfunction, intellectual disability or 'neurological burden' (subtle brain abnormalities & mild cognitive deficits), chronic/severe medical conditions, exposure to symptom models, and avoidant coping styles. Many of these factors are likely to be inter-related and might interact in contributing to the disorder.

There is evidence that DS may be precipitated by: acute increases in perceived stress, adverse or otherwise disruptive life events, and acute psychopathological disorders. In addition, perpetuating factors might include ongoing psychopathology or life stresses (e.g. dysfunctional relationships), avoidant coping strategies, and maladaptive

cognitive representations (particularly illness-related). Less is known about the processes that trigger individual seizures, although intense emotion, stressful situations, and dissociation of mental or somatic processes are likely to play a role in many patients.

There are many challenges to overcome in conducting research on the aetiology of DS. One of the core challenges is that of identifying an appropriate control group. Furthermore, comparison groups must be matched carefully on variables relevant to the study design. Many studies do not blind investigators or participants to diagnostic category, so there is often a risk of investigator and participant bias on research findings. Moreover, cross-sectional research designs are used frequently, which make the direction of causality difficult to ascertain (Fiszman et al., 2004). Methodologically, longitudinal, prospective studies might overcome some of these limitations (Sharpe & Faye, 2006), although these might be difficult to implement.

In addition, across studies investigating the same variables (e.g. trauma, stressful life events, psychopathology), a variety of measures have been used and in some cases, standardised measures are not included. Many studies are retrospective in design and based on analysis of patients' records, with variables often not measured in a systematic or explicit way. Small sample sizes can also increase the risk of Type 2 errors. Some research groups have included patients with mixed ES and DS, whereas others include patients with DS only, making interpretation complex.

Furthermore, many studies adopt the criterion of video-EEG evidence for inclusion of patients, which might introduce bias into the samples recruited. In some locations, video-EEG is not widely available due to the expense and technology required. Moreover, many suitable research participants might be unnecessarily excluded from studies due to having received the diagnosis through expert opinion or other diagnostic procedures. Given that DS is a (relatively) rare condition, and analyses are increasingly focusing on subgroups of patients, the video-EEG criterion may not be entirely pragmatic in all studies. There is also a possible inherent bias in recruiting patients from specialist neurology/epilepsy services, as mentioned in section 2.2.1. Previous studies often include patients with varying length of seizure disorder

(chronicity), which may be an additional confound. Patients who have experienced DS for a prolonged period of time may represent a particularly complex subgroup, differing on a number of psychological variables to those with DS of shorter duration.

When investigating the various risk factors for DS, it is important to take account of the baseline rates of these factors in the general population. Reuber (2008) also suggests the importance of identifying the nature of the relationships between risk factors and DS more specifically (i.e. examining which particular aspects of the risk factors influence the development of DS over others). Furthermore, the effects of specific risk factors on the development of DS may be mediated by other important ('third') variables. For example, the experience of trauma/abuse or difficult childhood circumstances (e.g. family dysfunction) may influence the development of DS through changes in affective processing style. In order to assess such possible influences, these potentially mediating psychological variables should be measured and included in research on the disorder.

## **2.5.Theoretical perspectives and models**

### **2.5.1. Historical perspectives**

'Hysterical' symptoms including convulsions, have been documented since the early texts of ancient Egypt and Greece, with the 'wandering uterus' as the suggested causal mechanism (D.N. Black et al., 2004; Isaac & Chand, 2006; Kozłowska, 2005; Tasca et al., 2012; Trimble, 2010). Emotionally-caused seizure-like events were also recorded in ancient Babylonian culture (Francis & Baker, 1999; LaFrance & Schachter, 2010). Moreover, hysterical phenomena were described during the middle ages, but often attributed to witchery (Trimble, 2010). However, Jean-Martin Charcot (1825-1893) brought the phenomena of hysteria back into the domain of medicine during his work at La Salpêtrière during the nineteenth century, with the "grande attaque" being considered the most extreme hysterical symptom (Faber, 1997).

Following Charcot, his student Pierre Janet systematically developed the concept of dissociation as a narrowing of the range of conscious awareness, and proposed this



mechanism as the underlying process of hysterical symptoms, linked to previous traumatic experiences and 'fixed ideas' (van der Hart & Horst, 1989). Janet's contemporary and fellow student of Charcot, Sigmund Freud described the concept of conversion, along with Josef Breuer (R.J. Brown, 2004). Freud proposed that hysterical symptoms represent psychic 'energy' converted into a symbolic physical symptom from unconscious emotional conflicts linked to previous traumatic experiences (V. Bell et al., 2011; D.N. Black et al., 2004; Trimble, 2010). These conflicts or unacceptable emotions were said to be 'repressed' into the unconscious, in order to regulate the conscious awareness of negative affect (Nicholson, Stone, & Kanaan, 2011). Psychoanalytic accounts of conversion have had a clear influence on contemporary conceptualisations of DS.

### **2.5.2. The role of trauma and emotion**

Following a psychodynamic approach, Betts and Boden (1992b) proposed that at least some types of DS are directly caused by previous sexual abuse. For example, they suggested that an 'abreactive' type of seizure (i.e. stiffening, uncoordinated jerking, and pelvic-thrusting, back-arching) was 'acting out' a flashback of the traumatic events. Furthermore, the authors also linked the 'swoon' type of attack (i.e. collapse, unresponsiveness, limpness) to a dissociative state, allowing the patient to 'switch off' from painful memories or flashbacks from their traumatic past.

Bowman (1993) proposed four different psychodynamic 'pathways' leading to the development of DS. The most common of these involved patients with a history of childhood abuse, who exhibited considerable dissociative symptoms. The onset of DS in such patients was often precipitated by a traumatic/stressful life experience that was proposed to be reminiscent of the original traumatic experience(s). A second pathway involved rape during adulthood, either with no childhood abuse or in addition to childhood abuse. In these patients, the DS were argued to be symbolic of the assault. Thirdly, some patients did not report traumatic experiences per se, but instead had experienced multiple stresses that were said to have exceeded their coping capacity. A final pathway was described as misdiagnosed panic attacks, some of which occurred after sexual trauma.

Additionally, Bowman and Markand (1996) reported that traumatic experiences were often rather remote in their sample, but that the onset of seizures was frequently precipitated by a recent stressful/traumatic experience that was in some way linked to the causal events. Bowman and Markand (1999) further proposed that precipitating events lead to DS via four emotional mechanisms, including: provoking affect linked to remote trauma/abuse, increasing anger/frustration to an intolerable extent, triggering sadness or anxiety regarding possible losses, and stimulating affect linked to unspeakable or insoluble interpersonal conflicts.

In summary, Bowman (2010) proposes that, for many patients, a combination of multiple stresses and traumas interact in overwhelming the individual's emotion regulation abilities. Furthermore, these emotion regulation abilities are argued to be weakened by the dysfunctional family contexts in which they develop.

### **2.5.3. Learning theory**

DS have been conceptualised from the perspective of learning theory by some authors. This approach suggests that DS are learned behaviour patterns, acquired through operant and social learning mechanisms, such as positive and negative reinforcement and symptom modelling (Bautista et al., 2008; Sirven & Glosser, 1998; Volow, 1986). Positive reinforcement could include the 'primary gain' of direct relief from unpleasant emotions such as anxiety (Moore & Baker, 1997; Ramani et al., 1980). Furthermore, negative reinforcement might be received in the form of avoidance or removal of undesired situations or demands (Baker et al., 1995; Frances et al., 1999; Ramani et al., 1980; Sirven & Glosser, 1998). It has also been proposed that the adoption of the role of an individual with a chronic illness (i.e. the 'sick role') confers considerable 'secondary gain', whereby the individual is able to avoid undesirable demands or experiences (Slavney, 1994), or receives financial and social benefits, such as governmental financial assistance or attention/nurturance from significant others (Baker et al., 1995; Volow, 1986).

Contemporary authors typically discuss learning/conditioning processes as shaping factors in the disorder, that allow symptoms to take the form of seizure-like episodes rather than a different form (e.g. Salmon et al., 2003). Modelling of the symptoms of

DS could take place through observation of others' seizures (e.g. family members with ES or DS), media representations of epilepsy, input from clinical epilepsy services (e.g. patients' information literature, clinicians' questions, diagnostic tests), and possibly modelling of one's own seizures in the case of those with comorbid ES (Volow, 1986; Oto & Reuber, 2014).

#### **2.5.4. Family dynamics**

Another area that has been discussed with reference to the aetiology of DS is that of family systems. Differences in areas such as emotional expression, control, and cohesiveness in the families of patients with DS may affect the development of emotional regulation and expression, and increase the tendency to experience psychological distress in physical ways (i.e. somatisation), or detach from difficult emotions (e.g. dissociation). Salmon et al. (2003), for example, argued that DS might be a manifestation of a need for greater control, in situations where the individual feels powerless to exert their needs or wishes (Salmon et al., 2003). The authors proposed a model in which poor parenting or adverse family dynamics may combine with abusive experiences to increase the risk of experiencing MUS in general.

Furthermore, Quinn and colleagues (2010) noted that many psychotherapists experienced in DS saw the seizures as a means of expressing emotion. This was viewed as a result of developing within dysfunctional interpersonal contexts (family systems) in which verbal communication of affect was censured, alongside encouragement/modelling of nonverbal affective expression. An important role for disrupted attachment has also been proposed in the aetiology of DS (Holman et al., 2008; Quinn, Schofield, & Middleton, 2008)

#### **2.5.5. Cognitive perspectives**

The core idea underlying cognitive accounts of conversion disorder/MUS is that the brain processes information in a hierarchical manner, with much of the basic motor and perceptual processing occurring low down in the system, relatively automatically, and below the level of conscious awareness. It is argued that MUS (and DS) could be the result of such processes occurring in the lower parts of the cognitive system (Carson et al., 2012).

Hilgard's neodissociation theory (1994) has had considerable influence on conceptualisations of dissociative and conversion processes. Based on hypnotic phenomena, Hilgard proposed the presence of a 'hidden observer' with access to knowledge and information not consciously available to the hypnotised individual. The theory proposed the existence of a central control or 'executive' structure that plans, monitors, selects and controls the actions of subordinate cognitive/behavioural systems. Once selected, these subordinate systems have some degree of autonomy and independence of functioning, and they exist within a hierarchical structure whereby any one may be dominant at a given time. The subordinate control systems include sets of specific routines/schemata, developed on the basis of previous experience. Hilgard argued that, under certain conditions, disruption to the connections between the central structure and the subordinate systems manifest in a loss of voluntary control/awareness of processes (e.g. memory, physical) carried out by the lower-level systems. In other words, the actions and operations of the subordinate systems have been 'dissociated' from the executive control system.

Subsequently, Oakley (1999) presented a cognitive model which sought to explain hypnotic, conversion and dissociative phenomena. Oakley differentiated between evolutionarily 'older' subcortical neural systems that mediate relatively automatic behaviours and more recently developed 'consciousness' systems (i.e. the neocortex). According to this model, the consciousness systems include a 'priority action system' (PAS), in which processes of relevance to the most urgent challenges are prioritised for more elaborate processing. The contents of the PAS are determined by an 'executive' structure that is also within the consciousness systems, but that is separate from the PAS. This executive structure was thought to be based in frontal neural systems and recruited when habitual or well-learned responses are not suitable, such as when confronted with novel situations or stimuli.

Oakley makes the key distinction that we are only currently aware of the outcomes of the processes occurring in the executive system, but not the processes themselves. It is the material that is selected by the executive system that constitutes our subjective experience (i.e. the contents of the PAS), not the process itself. According

to this model, it is possible to 'fool' the PAS into altered or aberrant subjective experiences, by inhibition of information flow into it, or by exaggeration or erroneous information being fed in. In addition, either the executive system or internal/external stimulation can initiate behaviour without involvement of the PAS. This model could be applied to explain the symptoms of DS.

R.J. Brown (2004; 2006) also proposed a cognitive account of MUS. According to this perspective, MUS are caused by automatic (preconscious) selection of the 'wrong' information for cognitive processing and automatic behavioural control by a primary attentional system (PAS). In other words, erroneous information is selected at the stage of attentional allocation. These erroneous 'hypotheses' are labelled 'rogue representations' and can be learned via direct and indirect exposure to physical states in the self and others, verbal suggestion and cultural knowledge about illness, bodily states and health.

More specifically, R.J. Brown argues that DS might occur as a result of environmental or internal cues automatically activating a procedural representation or schemata, characterised by seizure-like behaviour, perceptions and cognitions. The chronicity of MUS is thought to relate to repeated direction of attentional resources to the symptoms, which serves to increase the general level of activation in these 'rogue representations', thereby increasing the likelihood that such representations will be selected by the PAS automatically on an ongoing basis. Crucially, because these processes occur below the level of conscious awareness, the individual is not aware of the falsity of the interpretation, and thus fully believes in the reality of the symptoms experienced. Finally, this model attempts to take into account the influence of traumatic experiences. It is argued that traumatic experiences increase the likelihood of bodily-focused attention as this provides a means of reducing attention to the distressing emotions and thoughts linked to such events. Attentional resources are thus removed from self-regulation and instead allocated to bodily symptoms.

Within cognitive accounts, MUS (and thus DS) are conceptualised as disruption of normal psychological processes, rather than as entirely pathological phenomena (Halligan, Bass, & Wade, 2000). However, these cognitive accounts do not fully explore

the issue at the heart of conversion disorder (and dissociative disorders including DS): the central role of negative affect. Furthermore, Carson et al. (2012) recommend that such cognitive models are integrated with other theoretical perspectives, such as neurobiological accounts, and those including attachment, emotional and relationship problems.

#### **2.5.6. Neurobiological accounts**

There have been a number of valuable reviews of neurobiological findings in patients with conversion disorders and/or DS. Together, these reviews suggest that differences in pathways between several brain regions may play a role in DS and related conversion disorders (e.g. movement disorder), including regions of importance for emotion and its regulation (e.g. ventromedial prefrontal cortex, amygdala, anterior cingulate cortex), motor planning, execution and coordination (e.g. basal ganglia, motor cortex, cerebellum), cognitive/behavioural/motor control (e.g. dorsolateral prefrontal cortex) and areas involved in awareness of self and the environment, such as parietal cortex (Nicholson & Kanaan, 2009; Nowak & Fink, 2009; Perez et al., 2014; Spence, 2006; Vuilleumier, 2014). However, it has been noted that at present, there is too little evidence to propose a definite neurobiological model of conversion disorder (Carson et al., 2012; Nicholson & Kanaan, 2009).

#### **2.6. Conclusions**

Individuals diagnosed with DS are a complex and heterogeneous group. There are biological, psychological, and social/environmental contributions to the aetiology of the condition, with possibly more than one pathway. However, there remain large gaps in current understanding of the disorder. Considerable knowledge has accumulated regarding the general psychological characteristics of patients with DS; however, there has been much less research on the specific psychological mechanisms underlying the occurrence of the seizures. Experimental psychological research methods could be utilised more extensively to provide further insight into these psychological processes. In accordance with this proposition, Chapter 3 outlines the rationale and aims of the research subsequently presented in the thesis.

### **Chapter 3. Dissociation, emotion and dissociative seizures**

The current chapter provides a discussion of the concept of dissociation, and how this psychological process might relate to DS. Specifically, it is proposed that a dissociative mechanism might underlie the occurrence of DS. The possible contributions of emotional processes to DS are also discussed, and a literature review of previous research pertaining to this is presented. On the basis of the literature reviewed in the thesis so far, an aetiological framework is proposed, and a hypothetical model presented, aiming to combine affective, dissociative and cognitive accounts of DS. Finally, the rationale, aims, and hypotheses of the current research are provided.

#### **3.1. The nature of dissociation**

##### **3.1.1. Definitions and conceptualisation**

Stone (2006) succinctly defines dissociation as a “...disconnection of bodily perception, thoughts, emotions, memories and identity” (p.309). However, the term has previously been used to refer to a variety of inter-related processes or constructs (R.J. Brown, 2002; E.A. Holmes et al., 2005; Kennedy et al., 2004). Cardeña (1994) provided a useful summary of some of the different applications of the term ‘dissociation’. According to this account, there are three predominant ways in which the term has been conceptualised, including two different types of dissociation (1 and 2) and a proposed function (3), as follows:

1. dissociation as *non-integrated mental modules/systems* (e.g. dissociative amnesia, dissociative fugue, dissociative identity disorder, conversion disorders)
2. dissociation as an *alteration in consciousness* involving a feeling of separation from the self or the environment (e.g. depersonalisation, derealisation)
3. dissociation as a *defence mechanism* (i.e. serving to reduce emotional distress)

Moreover, two superordinate forms of dissociation have been proposed by E.A. Holmes et al. (2005). Detachment was proposed to involve the experience of “...an altered state of consciousness characterised by a sense of separation from certain

aspects of everyday experience, be it their body...their sense of self...or the external world” (p.5). This definition incorporates phenomena such as depersonalisation, derealisation, out-of-body experiences, and emotional numbing/blunting. On the other hand, compartmentalisation was argued to be characterised by “...a deficit in the ability to deliberately control processes or actions that would normally be amenable to such control...” (p.7), as in the case of conversion disorders or dissociative amnesia, for example. It was argued that the compartmentalised processes continue to operate, and are able to exert an influence on behaviour, emotion and cognition, albeit at a preconscious level. Dissociative Identity Disorder (DID) is an example of a disorder involving severe and enduring compartmentalisation.

Other authors have distinguished another sub-type of dissociation, namely somatoform dissociation. This term refers to “...phenomena that are manifestations of a lack of integration of somatoform experiences, reactions, and functions” (Nijenhuis, 2001, p.9). Thus, somatoform dissociation refers to the phenomena typically observed in somatoform disorders, such as conversion disorder. Somatoform dissociation might represent a subtype of compartmentalisation (R.J. Brown, 2004).

### **3.1.2. Pathological dissociation**

In the late nineteenth century, Pierre Janet proposed that psychological dissociation (*‘désagréations psychologiques’*) is a pathological phenomenon, involving a disruption of the mind’s usually integrated processes, manifesting in particular processes being isolated or ‘split off’ from conscious experience whilst simultaneously exerting control or influence over behaviour (R.J. Brown, 2002; 2004; Buhler & Heim, 2009; van der Hart & Horst, 1989;). Janet’s perspective suggested that such tendencies towards dissociation are in some respect ‘constitutional’, in other words, a weakness or flaw in the individual’s personality (R.J. Brown, 2002; Nicholson et al., 2011).

A distinction between non-pathological and pathological dissociation has been made more recently (e.g. Rodewald et al., 2010; Spitzer et al., 2006; N. Waller, Putnam, & Carlson, 1996). It is recognised that some dissociative experiences are fairly common in the general population (Gershuny & Thayer, 1999; Stone, 2006), and that dissociation occurs on a continuum from relatively ‘normal’ daily experiences (absentmindedness,



absorption) to some of the more severe identity disturbances that characterise dissociative identity disorder, at the other end of the spectrum (Bernstein & Putnam, 1986). Nemiah (1981, as cited by Foa & Hearst-Ikeda, 1996) proposed that pathological dissociation may have two key features: an altered experience of personal identity and amnesia for events occurring during a dissociative episode. Moreover, N. Waller and colleagues (1996) argue that pathological dissociation can be seen as a 'discrete latent variable' (p.301), a 'taxon', that is qualitatively different to non-pathological dissociation and is relatively rare in the general population.

### **3.1.3. Methods of investigating dissociation**

A wide variety of tools have been used to assess dissociative experiences and disorders, most commonly self-report questionnaires and structured clinical interviews. Self-report measures of 'trait' dissociative tendencies include the Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986), the Dissociation Questionnaire (DIS-Q; Vanderlinden et al., 1991), and the Multiscale Dissociation Inventory (MDI; Briere, 2002). These measures examine dissociative experiences that occur on an ongoing basis in daily life. However, there are also a number of self-report measures that assess 'state' dissociation, including the Peri-traumatic Dissociative Experiences Questionnaire (PDEQ; Marmar, Weiss, & Metzler, 1997), and the Clinician Administered Dissociative States Scale (CADSS; Bremner et al., 1998). State dissociation refers to immediate dissociative experiences occurring at a given time-point.

Moreover, a self-report measure of somatoform dissociation has also been created (Somatoform Dissociation Questionnaire, SDQ; Nijenhuis et al., 1996), which has been developed in a short-form (5 items, SDQ-5; Nijenhuis et al., 1997) and a longer form (20 items, SDQ-20; Nijenhuis et al., 1996). On the other hand, the Structured Clinical Interview for DSM Dissociative Disorders (SCID-D; Steinberg et al., 1993) is thought to be less susceptible to social desirability biases than self-report measures, and also allows diagnosis of dissociative disorders where present. However, a highly qualified professional is required to administer the SCID-D and the interview is relatively time-consuming; therefore, it may be less efficient than self-report measures and impractical in some research contexts.

### **3.1.4. Biological correlates of dissociation**

Whilst dissociation is not thought to be caused by a specific organic pathology, it is accepted that functional neurobiological differences might be associated with the phenomena (Kihlstrom, 2005). Dissociative states might be triggered by high levels of autonomic arousal, such as during an acute traumatic event (Krystal et al., 1996; Marmar et al., 1998). However, the pathophysiology of dissociation remains poorly understood (Krystal et al., 1996; van der Kruijs et al., 2011). Investigators are now starting to attempt to investigate neurobiological processes correlated with dissociative and conversion phenomena using functional neuroimaging. A number of studies have found functional neural differences in patients with dissociative disorders (e.g. Ghaffar, Staines, & Weinstein, 2006; Staniloui et al., 2012; Stone et al., 2007; van der Kruijs et al., 2012).

## **3.2. The causal link between trauma and dissociation**

### **3.2.1. Trauma as a risk factor for elevated dissociation**

Elevated (somatoform and psychological) dissociative symptoms and pathological dissociation have been repeatedly found to be associated with previous traumatic experiences, particularly childhood abuse. Studies have reported this relationship in both clinical and non-clinical samples (Anderson, Yassenik, & Ross, 1993; Boysan et al., 2009; Briere et al. 2006; Chu & Dill, 1990; DiTomasso & Routh, 1993; Draijer & Langeland, 1999; Engel et al., 1996; Foote et al., 2006; Gershuny & Thayer, 1999; Goodwin & Sachs, 1996; Irwin, 1996; 1999; Isaac & Chand, 2006; Modestin et al., 1996; Mulder et al., 1998; Nijenhuis, Spinhoven, et al., 1998; Nijenhuis, van Dyck, et al., 2003; Roelofs et al., 2002; Ross & Ness, 2010; Sack, Boroske-Leiner, & Lahmann, 2010; Sar et al., 2000; 2007; G. Waller et al., 2001).

Briere (2006), for example, reported that 90% of individuals from the general population with clinical elevations on one or more dissociation scales (on the MDI) had a history of trauma. Moreover, a study of psychiatric inpatients (Draijer & Langeland, 1999) indicated that sexual abuse and physical abuse were highly predictive

of elevated dissociation scores. Similarly, Nijenhuis, Spinhoven, et al. (1998) reported that physical and sexual trauma were predictive of somatoform dissociation, and sexual trauma was predictive of psychological dissociation, in psychiatric patients. Furthermore, Anderson and colleagues (1993) reported that 88.2% of a sample of female sexual abuse survivors met the criteria for a dissociative disorder in adulthood. These are just a small selection of studies that have indicated a strong association between traumatic life events, elevations in dissociation and/or the presence of dissociative disorder. However, Kihlstrom (2005) noted several methodological limitations in this research literature, such as retrospective assessment of variables, uncorroborated reports of trauma/abuse and lack of clinical control groups in many studies. Future studies should aim to address these limitations.

### **3.2.2. Peri-traumatic dissociation**

Peri-traumatic dissociation is a well-documented phenomenon (Marmar et al., 1998), and provides one of the most compelling examples of the direct causal link between traumatic experiences and dissociation. Peri-traumatic phenomena can include derealisation and depersonalisation (e.g. altered sense of time, out-of-body experiences), but also somatoform phenomena such as altered vision, pain perception or motor control. Therefore, symptoms characteristic of both psychological and somatoform dissociation can occur in close proximity to traumatic events.

During, or in the short-term aftermath of a traumatic experience, dissociation may well be an adaptive mechanism (R.J. Brown, 2002), by which the individual can continue functioning or responding in an adaptive manner, despite extremely intense negative affect. In fact, symptoms of somatoform dissociation, such as motor inhibition and analgesia, have been proposed to serve a similar function to the typical animal defensive reactions observed in response to direct threat and/or injury, such as 'freezing' (Nijenhuis, 2001; Nijenhuis, Vanderlinden, & Spinhoven, 1998). Nevertheless, peri-traumatic dissociation is thought to be a risk factor for the later development of PTSD (Breh & Seidler, 2007; Ozer et al. 2003).

### **3.2.3. Dissociation in PTSD**

Dissociative symptoms are among the core symptoms required for a positive

diagnosis of PTSD, including emotional numbing/constriction, 'flashbacks', and detachment. Indeed, a key conceptualisation of PTSD is that the traumatic experiences have been dissociated from consciousness, potentially accounting for subsequent intrusive memories (Chu, 1996). In fact, some authors have proposed the existence a distinct subtype of PTSD, characterised by elevated dissociative symptoms, which could represent a form of 'emotional over-modulation' (Lanius et al., 2010, p. 640).

#### **3.2.4. Theoretical perspectives: trauma and dissociation**

Janet articulated clear links between hysteria/dissociation and traumatic experiences (Buhler & Heim, 2009), proposing that traumatic experiences can trigger the 'narrowing of the field of consciousness'. Janet further suggested that traumatic memories can remain in the mind in the form of subconscious 'fixed ideas' (Brown & van der Hart, 1998), which can be activated by external stimuli, and might continue to influence behaviour and experience.

Furthermore, it has been argued that childhood trauma is specifically linked to higher levels of dissociation in adulthood, because the child more readily adopts a dissociative style of coping with emotionally overwhelming events (Chu, 1996). Sack and colleagues (2010) argue that early traumatising, particularly that of a sexual nature, can provoke a protective dissociative response in order to cope with the resultant psychological distress; this dissociative response manifests as "discontinuity between the self and body" (p.315). This discontinuity could become a habitual way of managing psychological or physical pain.

Putnam (1997) proposed a developmental model of dissociative psychopathology, in which repeated traumatising during infancy or childhood disrupts the usual development of integrative functions of the mind, with dysfunctional attachment relationships compounding this process. It was argued that these disruptions lead to the individual experiencing behavioural and mental states that are not integrated, otherwise known as dissociative states. This model is known as the 'discrete behavioural state' model.

Furthermore, Van der Hart and colleagues (2004) have proposed a model of trauma-related dissociation based upon the concept of 'structural dissociation of the personality' (p.907). According to this model, dissociation involves encoding trauma-related information in a 'dissociated part of the personality' (p.908), which would allow such information to be accessible in certain conditions only. The authors argued that maintenance of structural dissociation is largely due to learning processes, including associative fear conditioning, evaluative conditioning and social learning processes.

Dalenburg and colleagues (2012) conducted an extensive review of studies relevant to the trauma model of dissociation and concluded that there is a strong association between trauma and dissociative experiences (as a continuum), and between trauma and diagnoses of dissociative disorders. The authors also proposed that dissociation occurs as a regulatory mechanism in response to extreme negative affect, such as fear.

### **3.3. Dissociative seizures as dissociative phenomena**

As described in Chapter 1 (section 1.1), psychogenic seizures are classified as 'dissociative convulsions' in ICD-10. This term has been broadened to 'dissociative seizures' by some authors (e.g. Goldstein & Mellers, 2006; Schmutz, 2013), as the attacks can include phenomena that resemble ES that are not necessarily convulsive in nature (e.g. atonic episodes). Many experts now propose that DS are a manifestation of dissociated mental processes, in at least a proportion of cases (Baslet et al., 2010; R.J. Brown, 2002; Harden, 1997; Kuyk et al., 1996; Goldstein & Mellers, 2006; Mellers, 2009; Moore & Baker, 1997; Roberts & Reuber, 2014).

#### **3.3.1. The causal role of trauma in DS**

On the basis of elevated levels of trauma, particularly childhood abuse in patients with DS (see Chapter 2, section 2.2.1), authors have proposed a prominent aetiological role for traumatic experiences. It is possible that, in some cases, DS represent a manifestation of the dissociative subtype of PTSD (Fiszman et al., 2004; Reuber, Howlett, & Kemp, 2005). Furthermore, as proposed by Bowman (1993), when a history of trauma is not present, it is possible that multiple stressors might also predispose towards dissociative tendencies and/or DS. As suggested by Reuber and

colleagues (2005), the condition known as Prolonged Duress Stress Disorder may be of relevance to DS, as it is similar to PTSD in presentation, but thought to be caused by long-term stress during development, rather than acute/isolated traumatic events.

### **3.3.2. Dissociative phenomena in patients diagnosed with DS**

Stone and Carson (2013) described a case series including a number of patients with DS who seemed to make a conscious choice to dissociate at the time of their seizures, termed 'wilful submission' to seizure onset. Furthermore, as noted by Mellers (2009), many patients with DS anecdotally report feeling disconnected from their surroundings during seizures. Peri-ictal symptoms of depersonalisation/derealisation have also been found to be elevated in patients with DS relative to those with ES (Hendrickson et al., 2014).

Rates of comorbid dissociative disorders are often high in samples with DS (Baillés et al., 2004; Bowman, 1993; 2010; Bowman & Markand, 1996; Litwin and Cardeña, 2000; Marchetti et al., 2009). Patients diagnosed with DS have also been found to receive elevated scores on measures of trait dissociation such as the DES (Bowman, 1993; Mitchell et al., 2012). Furthermore, several studies have reported higher DES scores in patients with DS relative to those with ES (Akyuz et al., 2004; Dikel et al., 2003; Goldstein & Mellers, 2006; Ito et al., 2004; 2009; Mazza et al., 2009; Proença et al., 2011; Prueter et al., 2002; Reuber, House, et al., 2003; van der Kruijs et al., 2012; van Merode et al., 2004) and healthy controls (Goldstein et al., 2000; Ito et al., 2004; 2009; Mazza et al., 2009; O'Brien et al., 2015). However, DES scores have been reported to be comparable in patients with DS only and those with mixed ES and DS (Mitchell et al., 2012). Interestingly, one study showed that DES scores were significantly higher in traumatised patients with DS, compared to those who did not report traumatic life events (Hingray et al., 2011).

On the converse, some investigators have failed to find significant elevations in DES scores for patients with DS compared to those with ES (Alper et al., 1997; Bowman & Coons, 2000; Fleisher et al., 2002; Litwin & Cardeña, 2000). Nonetheless, using factor analysis, Alper et al. (1997) reported that their DS group showed significantly higher scores than the ES group on a factor described as 'depersonalisation-

derealisation', and Bowman and Coons (2000) found that the patients with DS scored significantly higher on the SCID-D, relative to those with ES. Moreover, the studies of Fleisher et al. (2002) and Litwin and Cardeña (2000) both had methodological limitations, namely the inclusion of patients with comorbid ES in the DS group, and a very limited sample size (DS  $n = 10$ ) respectively.

When other scales have been used to measure dissociation in patients with DS, findings have been mixed. Ozcetin and colleagues (2009) and van der Kruijs et al. (2012) reported significantly higher scores on the DIS-Q in patients with DS relative to healthy control participants; however, Kuyk et al. (1999) failed to observe higher DIS-Q scores in DS patients relative to those with ES. Interestingly, scores on the CADSS did not differ between groups (DS, ES) in one study, despite significant differences on the DES (Akyuz et al., 2004). This finding highlights the importance of assessing dissociative phenomena using more than one measure, if possible. The CADSS measures state dissociation, in contrast to the DES which assesses trait dissociative symptoms.

A number of studies have also examined the presence of somatoform dissociation in patients with DS. Kuyk et al. (1999), for example, found that somatoform dissociation discriminated between DS and ES groups, although psychological dissociation did not. Higher scores on the SDQ-20 in DS patients relative to those with ES have also been reported in other studies (R.J. Brown et al., 2013; Lally et al., 2010). Furthermore, patients with DS have been shown to receive higher SDQ-20 scores compared to healthy control groups (van der Kruijs et al., 2012; Xue et al., 2013). Spinhoven et al. (2004) observed elevated SDQ-20 scores in patients with DS, relative to normative samples. Despite this, Bodde et al. (2013) reported that DS patients did not deviate from normative scores on either the SDQ-20 or the DIS-Q. Two subtypes of dissociation were examined by Lawton et al. (2008), in patients with DS and ES. These were depersonalisation ('detachment') and somatoform dissociation ('compartmentalisation'). Whilst the DS group reported significantly elevated symptoms of the latter form of dissociation (relative to ES patients), this finding did not survive statistical correction for symptoms of anxiety and depression.

Regarding comparisons of patients with ES and DS on dissociation measures, as noted by other authors (R.J. Brown, 2002; Lawton et al., 2008), some phenomena that occur during ES are superficially similar to dissociation, but many of them are not actually dissociative because they are caused by epileptogenic electrophysiological activity. Nevertheless, the superficial resemblance of ES symptoms to dissociative phenomena is likely to confound scores on measures such as the DES; therefore, comparing patients with ES and DS on such measures may not be informative.

In summary, the current evidence generally supports the proposition that patients with DS experience elevated dissociative symptoms, relative to patients with ES and healthy controls. The mixed findings are most likely due to methodological weaknesses, such as imprecise measures that do not differentiate different types of dissociation, inclusion of comorbid ES in DS samples, limited sample sizes, and lack of control for possible confounds (i.e. general psychological distress).

### **3.3.3.Theoretical perspectives on dissociation and DS**

Several authors have proposed a shared mechanism underlying the dissociative and conversion disorders, and MUS more generally. Kihlstrom (2005, p.242), for example, notes the common feature of “...divisions in consciousness, and dissociations between explicit and implicit memory and perception...”. Bowman and Markand (1996) propose that dissociative, somatoform and conversion disorders may represent a spectrum of dissociative-somatic reactions to trauma. In addition, V. Bell et al. (2011) conceptualised conversion/hysteria as a specific type of dissociation (compartmentalisation), entailing disconnection between elements of cognition and conscious awareness/control. Similarly, Harden (1997) points out that both dissociation and conversion refer to phenomena over which the individual has no voluntary control or awareness.

As mentioned in Chapter 2 (section 2.5.5), R.J. Brown has presented a cognitive account of MUS (2004; 2006); however, he also applied this perspective to DS specifically (2002). According to this perspective, DS may represent a previously learned behavioural routine, triggered by environmental cues without conscious awareness or executive control. A cognitive control system was proposed to have a



key role in inhibiting the executive system and the information feedback from lower level systems. This control system was proposed to be directly activated by environmental triggers. The inhibition of executive control occurs after the activation of lower-levels subsystems, rather than before, in this model. R.J. Brown (2002) argued that the behavioural routines could be learned through symptom modelling, or that they could be an innate and evolutionarily old defensive function. In terms of environmental cues, triggers were proposed to include any stimuli associated with the learned behavioural routines, in addition to anxiety and/or associated thoughts, images or memories. This theory proposes an intuitively and theoretically plausible account of DS; however, it does not explain in detail how anxiety or other intense affect might activate the cognitive control and behavioural systems.

Not all authors agree that a dissociative account of DS applies to all cases. Roberts and Reuber (2014), for example, assert that a dissociative mechanism only applies to a proportion of patients. Schmutz (2013) also proposes that there are several subgroups of patients exhibiting DS, with different underlying psychological causation including somatoform dissociation, psychological dissociation, PTSD, conversion disorder, and adjustment disorders. On the other hand, Bodde et al. (2009) and Bowman (2010) argue that dissociation is not the cause of DS, but instead is the psychological mechanism underling the seizures.

#### **3.3.4. Summary and conclusions: trauma, dissociation and DS**

Dissociation has been closely linked to traumatic and highly stressful life experiences, particularly those occurring in childhood. It is possible that individuals subjected to severe or repeated trauma or multiple stressors during development may develop a habitually dissociative way of processing information and responding to stress, which can be termed 'trait' dissociation. On the other hand, 'state' dissociation might serve to reduce the short-term emotional impact of an acute trauma, highly stressful event or intolerable levels of affective distress/arousal. The presumed function of both trait and state dissociation is to reduce the subjective experience of intense or aversive emotional states.

Patients with DS often report trauma and/or multiple stressors in their histories. An increasingly accepted perspective on the nature of DS is that they are a manifestation of a dissociative psychological process. In the present thesis, it is proposed that dissociation, defined as an *involuntary reduction/loss of normal control or awareness of bodily or psychological processes*, constitutes the mechanism that is likely to underlie DS. Moreover, it is suggested that this mechanism is potentially shared with other dissociative and conversion/somatoform disorders. The next section explores how emotional processes might relate to an underlying dissociative mechanism in this disorder.

### **3.4. The role of emotion in dissociative seizures: previous theoretical perspectives**

Emotions are central to most influential theories of dissociation (Oathes & Ray, 2008). As previously mentioned, dissociation is commonly thought to be a response to, and/or an unconscious attempt to cope with (negative) affective experience; it may also reflect failures in the integration of affect (Rodin, de Groot, & Spivak, 2002). Rodin and colleagues (2002) suggest that the shared feature between trauma, dissociation and somatisation is that they all involve altered processing of emotions. As proposed by Roberts and Reuber (2014), one manifestation of dissociation could be a loss of the usual connections between different aspects of emotional processing (e.g. subjective, physiological), or reduced integration of emotional responses into current consciousness or identity.

Cutting across almost all theories of the aetiology of DS is the basic idea that an intolerance, avoidance, or difficulty coping with anxiety or emotional distress/arousal contributes to the occurrence of the episodes (Dimaro et al., 2014; Roberts & Reuber, 2014). For example, Moore and Baker (1997) suggested that DS are likely to be a manifestation of high levels of emotional arousal. Bodde et al. (2009) suggested that an increased tendency to dissociate is an 'emotional mechanism' (p.9) that serves to trigger the seizures (in some cases). Authors generally concur that the seizures serve to release, express or avoid intense or over-whelming emotional states (e.g. Bowman & Markand, 1999; Goldstein & Mellers, 2006; Rosenbaum, 2000).

Goldstein and Mellers (2006; 2012) have proposed a dissociative model of DS, suggesting that the episodes represent a “...dissociative response to emotional arousal.” (p.438). It was argued that during the seizures, patients experience autonomic signs of emotional arousal, which may be triggered by emotionally significant environmental cues, such as trauma-associated stimuli. However, alongside the elevated physiological arousal, subjective states suggest that conscious awareness of the arousal and associated emotion is impaired. This constitutes the basic mechanism by which DS may operate and may be the ultimate ‘reward’ for the patient – protection of the individual’s consciousness from the highly aversive affect. The authors recommend further investigation of the subjective phenomena experienced by patients during their seizures, alongside physiological measures of autonomic arousal.

Baslet (2011) more recently hypothesised that patients with DS may have reduced tolerance for arousal. It was proposed that specific triggers (sensory, emotional, cognitive) might cause patients' arousal levels to deviate from their narrow tolerable level, initiating the occurrence of DS symptoms. It was proposed that triggers might be difficult to identify in many instances, due to alterations in preconscious emotional information processing. He further argued that some of the autonomous behaviours observed during DS (e.g. autonomic arousal, movements) might be 'pre-wired' responses that are not accessible to cognitive/voluntary control, and only occur in the absence of appropriate cognitive processing. During DS, Baslet suggested that the cognitive, emotional and sensorimotor systems may be operating in an autonomous manner. Furthermore, it was proposed that emotional arousal may act as a core trigger in the initiation of individual DS, directly influencing cognitive control and behavioural manifestations. This mechanism was suggested to involve abnormal connectivity between 'emotion' areas of the brain (e.g. amygdala), the 'executive'/cognitive integration areas (e.g. lateral prefrontal cortex), and areas involved in automatic (pre-wired) behavioural tendencies (e.g. sensorimotor areas).

Furthermore, Roberts and Reuber (2014) also argued that emotion has a central contributing role in triggering DS. They proposed three mechanisms by which

emotional states might lead to DS. One is similar to the typical dissociative account of DS; that consciousness is reduced due to excessive inhibition of emotional processing. Secondly, that the behavioural symptoms and loss of consciousness in DS are direct manifestations of intense emotional experiences, with no dissociative mechanism occurring. Finally, they propose that minor fluctuations in emotion or affectively-conditioned innocuous stimuli may elicit DS preconsciously.

The dissociative mechanism proposed by Roberts and Reuber (2014) assumes the involvement of excessive inhibition of emotion. However, another possibility is that the emotional processes continue, yet it is just the awareness of these emotions that is inhibited. This would still represent a dissociative mechanism, but would explain why patients with DS frequently experience symptoms of emotional arousal, without subjective experience of the emotion. In the model proposed by Roberts and Reuber (2014), it is assumed that DS are distinguishable from the process of dissociation. However, it is proposed here that the seizure itself is a dissociative phenomenon; that is, a loss of integration between conscious awareness, voluntary control, and emotional experience. Many, if not all DS involve disturbances of responsivity, voluntary control and alterations of consciousness/awareness. These disturbances can be seen as dissociation in and of themselves.

### **3.5. Emotion in patients diagnosed with DS: literature review**

#### **3.5.1. Self-report measures**

##### ***General emotional distress***

As described in Chapter 2 (sections 2.2. and 2.3), patients diagnosed with DS experience considerable emotional distress on an ongoing basis. They often report elevated stress in daily life, in addition to numerous recent or remote adverse life events. Generally elevated symptoms of depression and anxiety are commonly reported in the literature. Self-injurious and suicidal behaviours are also documented in some cases. DS patients report elevations in fear sensitivity (Hixson et al., 2006), suggesting heightened anxiety levels in response to various stimuli and situations. Panic symptoms are also frequently reported in this group (e.g. Frolov et al., 2003;

Hendrickson et al., 2014; Silva et al., 2001; Witgert, Wheless, & Breier, 2005). Furthermore, one study reported heightened experience and expression of anger and hostility in patients with DS, in comparison with control participants (Mökleby et al., 2002). In summary, patients with DS seem to experience considerable emotional distress in daily life.

### ***Emotional dysregulation (ED)***

Numerous studies have indicated that patients with DS have difficulties in regulating their emotional states and reactions. Personality traits associated with emotional under-control (e.g. BPD) are the most common patterns reported. For example, Reuber, Pukrop, et al. (2004) found that patients with DS reported significantly greater trait ED than both healthy and ES control groups. Moreover, several research groups have explored subtypes of ED in this group. Bodde et al. (2013) reported that, relative to normative standards, patients with DS showed significant elevations on a number of subscales from the Cognitive Emotion Regulation Questionnaire (CERQ; Garnefski & Kraaij, 2007), which included 'acceptance' (tolerance of current emotion and acceptance of the negative experience), 'rumination' (focusing on the feelings and thoughts provoked by a negative experience), 'self-blame' (placing responsibility for events on the self) and 'positive refocusing' (channelling attention from the negative experience to more positive and rewarding stimuli or experiences). Obviously, not all of these emotion regulation strategies are necessarily maladaptive. One limitation of this study was the inclusion of a 'cognitive' measure of emotion regulation only (excluding behavioural strategies). Nevertheless, the findings suggest that patients with DS may have different ways of consciously regulating their emotions, when compared to the general population.

In addition, Gul and Ahmad (2014) reported that patients with DS scored higher on emotion suppression and lower on cognitive reappraisal than healthy control participants, as measured with the Emotion Regulation Questionnaire (Gross & John, 2003). Another sample of patients with DS scored comparably to healthy controls on the Affect Intensity Measure (Larsen & Diener, 1987), suggesting similarly intense emotional reactions between groups (Urbanek et al., 2014). However, this sample reported a greater tendency to control anxious or dysphoric (unhappy) affective

states and expressed more negative beliefs about emotions, compared to the control group.

On the other hand, some authors have identified subgroups of patients with DS who exhibit particular difficulties in ED. R.J. Brown and colleagues (2013) reported that only a subgroup of their sample of DS patients showed deficits in ED relative to patients with ES, including difficulties in identifying, accepting and describing feelings, carrying out goal-directed behaviour, controlling feelings/actions, and using adaptive regulatory strategies. The authors argued that this group could be characterised as showing an 'under-modulation of affect', with DS representing a mechanism by which intense emotional distress is interrupted and relieved. However, several limitations were also noted by the authors, including the use of a postal survey, small sample size, and a lack of assessment of comorbid psychopathology or neurological illnesses/injury.

Uliaszek and colleagues (2012) also identified two specific subgroups of patients with DS using cluster analysis. One subgroup showed a pattern of generally high levels of ED; however, another subgroup reported particularly high levels of emotional regulation, specifically with regard to awareness of emotional reactions and the ability to maintain goal-driven behaviour despite such emotional reactions. Furthermore, belonging to the high ED group significantly predicted a number of psychological variables (e.g. depression, anxiety, stress, dissociation). As might be expected, the group high in ED also had significantly more comorbid psychiatric diagnoses than the low dysregulation group.

Together, the findings suggest that a notable proportion of patients diagnosed with DS experience considerable ED, relative to normative standards and clinical control groups. High levels of ED seem to be linked to increased general emotional distress, dissociation and reduced quality of life in this group.

### ***Alexithymia***

Patients with DS have been found to score higher on measures of alexithymia than control participants (Bewley et al., 2005; Kaplan et al., 2013; O'Brien et al., 2015; Urbanek et al., 2014). However, findings have been inconsistent, with one study

(Urbanek et al., 2014) reporting elevations on all subscales of the Toronto Alexithymia Scale-20 (TAS-20; Bagby, Parker, & Taylor, 1994), and others finding no differences in alexithymia between DS patients and control groups (Myers, Fleming, et al., 2013; Tojek et al., 2000; Wolf et al., 2015). On the other hand, Kaplan et al. (2013) reported that reduced scores for 'identification of affective states' significantly predicted a diagnosis of DS (rather than ES), and there was a non-significant trend of the same nature for 'describing emotions'. Therefore, it seems that some aspects of alexithymia might be more prominent in patients with DS than others. In fact, Urbanek et al. (2014) reported that scores on the 'poor understanding of emotions' subscale of the TAS-20 was a highly significant predictor of a diagnosis of DS (relative to healthy control participants).

Urbanek et al. (2014) also reported that a significantly larger proportion of their DS group scored in the alexithymic range on the TAS-20 than in the control group. However, whilst a large proportion of the DS sample included by Bewley et al. (2005) were classified as alexithymic on the TAS-20 (90.5%), the overall scores did not differ significantly from control groups (ES, healthy controls) once the influence of anxiety and depression was accounted for statistically. Nonetheless, scores on two subscales were significantly higher in the DS group relative to the healthy control group, indicating worse identification and description of feelings. R.J. Brown and colleagues (2013) reported that only a subgroup of patients with DS were more alexithymic than patients with ES (those with higher levels of ED, general psychopathology, and somatisation). Hingray et al. (2011) found that scores on the TAS were high in DS patients with and without a history of trauma. Interestingly, the study by Myers, Fleming, and colleagues (2013) identified significant associations between alexithymia and arousal (anxiety), intrusive experiences, dissociation, and avoidance (defensive) in the DS group.

As yet, therefore, the findings are rather mixed regarding alexithymia in patients with DS. Nonetheless, the current evidence points towards reduced proficiency at identifying, describing and understanding emotions in patients with DS. Moreover, these difficulties seem to be linked to other important characteristics in this group, such as ED, general psychopathology and dissociation/somatisation.

### ***Peri-ictal affect***

A notable proportion of patients with DS report emotional triggers as precipitants of individual DS (Lempert & Schmidt, 1990). Moreover, many patients experience symptoms suggestive of anxiety, fear/panic and/or autonomic arousal prior to or during their seizures (Goldstein & Mellers, 2006; Hendrickson et al., 2014; Moore & Baker, 1997). The findings of Goldstein & Mellers (2006) suggested elevated somatic anxiety symptoms in DS patients (compared to those with ES), in the absence of increased subjective anxiety. This is indicative of some form of dissociation of physiological and conscious emotional experience.

### ***Evaluation of self-report studies***

Many of the limitations of self-report questionnaires have been discussed in previous sections/chapters. One important weakness of such methods of relevance to emotional processes is that they only allow responses relating to processes of which patients are consciously aware. Given that the psychological mechanisms underlying the symptoms of DS are presumed to take place below the level of conscious awareness, such mechanisms, by definition might be unamenable to examination using introspection (i.e. self-report).

### **3.5.2. Experimental studies**

To date, there have been only a handful of published articles describing experimental or standardised investigation of affective processing in patients with DS. One study incorporated a test of affect recognition and expression within a standardised test battery (Barrow Neurological Institute Screen for Higher Cerebral Functions; Prigatano, Amin, & Rosenstein, 1995). The affective component included elements requiring verbal expression of affect, recognition of facial (pictorial) emotional expressions, responses to a humorous stimulus and general affect control during testing (Prigatano & Kirlin, 2009). Patients with DS received lower scores on this affective subtest compared to those with ES. Whilst this finding was indicative of deficits in affective processing, the findings did not indicate which aspects of the subtest had posed the greatest difficulty for the patients. Moreover, the test was not administered under controlled laboratory conditions.



Nonetheless, the same year, Bakvis, Roelofs, and colleagues (2009) carried out a laboratory-based experimental study of preconscious processing of emotional facial expressions in patients with DS, relative to healthy controls. A masked emotional Stroop paradigm was used, which included the preconscious presentation of either angry, neutral or happy faces. The faces and masks were presented in one of three colours, and participants were required to name the colour of the masking stimuli aloud. Attentional bias scores were calculated for the happy and angry faces separately, by subtracting the average reaction time for each from that of the neutral condition. This task was administered under a rest condition and when participants were under stress induced by the Trier Social Stress test (Kirschbaum, Pirke, & Hellhammer, 1993). Performance of the DS patients was compared to that of healthy controls, with no significant differences noted under the stress condition. However, under the rest condition differences emerged; DS patients had higher attentional bias scores for the angry faces compared to the healthy control group. The authors interpreted this as hypervigilance to facial anger (threat). These results are suggestive of a possible propensity for DS patients to allocate a disproportionate degree of attention to facial signals of threat, when in a resting state. Furthermore, this attentional bias was positively correlated with basal hypercortisolism and self-reported sexual traumatic experiences in the patients with DS. The strengths and limitations of this study are discussed in Chapter 6.

An additional study by the same research group (Bakvis et al., 2011) assessed 'approach-avoidance' behaviours towards happy and angry facial expressions, by measuring flexion or extension of participants' arms respectively. On this task, patients with DS were significantly slower in responding to angry facial expressions with the approach behaviour compared to avoidance behaviour; whereas, this effect was not evident in the control group. This finding was still significant when anxiety, depression and medication effects were controlled in the analysis. Furthermore, the increased reaction times for anger approach were positively correlated with baseline cortisol. The effect disappeared after stress induction with the Cold Pressor test (Hines & Brown, 1936). These findings suggested that, under resting conditions, the patients with DS showed a tendency towards behavioural avoidance of facial threat signals, and that this avoidance was linked to generally elevated stress levels. However,

a very small sample size was included in this study (DS  $n = 12$ ), and so replication in a larger sample might be beneficial.

Another study by Bakvis, Spinhoven, Putnam, et al. (2010) reported that at baseline (prior to stress-induction), patients with DS showed higher levels of interference by facial distractor stimuli on a working memory task, relative to healthy controls. It is possible that this finding could be linked to the general hypervigilance to social stimuli reported in previous studies, although the effect interference effect was across positive, negative and neutral faces in this study. In addition, Gul and Ahmad (2014) reported that in a task-switching paradigm (evaluation of emotion or age of faces), patients with DS had significantly longer reaction times for categorisation of facial age compared to facial emotion. Reaction times were longer when task-switching from the emotion to the age-discrimination task. Together, the authors interpreted these findings as an attentional bias towards the emotional (happy/angry) aspects of the faces. However, the relative effects of emotion categorisation for positive (happy) and negative (angry) faces were not examined separately in this study. Moreover, Gul and Ahmad (2014) excluded patients who were over the age of 35 years and those with comorbid disorders (except mild anxiety, depression and stress); therefore the generalizability of the findings are limited.

Another recent study (Dimaro et al., 2014) included the Implicit Relational Assessment Procedure (Barnes-Holmes et al., 2006), to assess implicit beliefs about anxiety in patients with DS. Reaction times towards consistent and inconsistent statements about anxiety-related states (i.e. worried, tense scared) or the opposite (i.e. calm, relaxed, secure) were measured. The three groups tested (ES, DS, non-clinical controls) did not differ on this measure; however, a key difference was that patients with DS had significantly greater discrepancies between their explicit and implicit anxiety levels, relative to both control groups. The discrepancy was characterised by higher explicit anxiety than implicit anxiety. However, it is unclear whether any screening criteria were used to recruit the non-clinical group, as no statement was made about exclusion criteria (e.g. medical conditions, substance use). Indeed, the clinical groups were also not described in detail; for example, no data on formal psychiatric history, length of seizure disorder, and/or use of medications was

provided.

Another pertinent study was carried out by Roberts and colleagues (2012), in which responses to affective pictures (IAPS; International Affective Picture System; Lang, Bradley, & Cuthbert, 2005) were investigated in patients with DS compared to non-clinical controls (high or low in post-traumatic symptoms; PTS). No between group differences were found for ratings of valence across the pleasant, unpleasant and neutral stimuli. However, patients with DS rated the pictures as significantly more intense than both control groups (high and low PTS). On closer inspection, there were no differences in ratings of emotional intensity for the unpleasant images. On the other hand, patients with DS reported more intense emotional responses to the positive images, relative to the control group who were low in PTS, but not relative to those high in these symptoms. For neutral images, however, the DS patients reported more intensity than both control groups. The finding of increased intensity ratings in patients with DS was interpreted by the authors as suggestive of increased attentional focus on physical manifestations of arousal. However, the lack of group differences on valence ratings indicated that patients with DS did not differ in the positivity/negativity of their subjective responses to the stimuli.

Whilst there were no group differences in cardiovascular measures of emotional responding to the stimuli, baseline respiratory sinus arrhythmia (RSA) was found to be lower in the DS group than in the PTS-low control group (Roberts et al., 2012). This is suggestive of decreased parasympathetic activity and, therefore, of higher levels of sympathetic activation at rest. This finding concurs with the basal hypercortisolism observed in a DS sample by Bakvis, Spinhoven, Giltay, et al. (2010). However, there were several methodological limitations to note in Roberts et al's (2012) study, some of which were discussed by the authors. One issue was the inclusion of a modest sample size ( $n = 18$  in each group). This may have reduced statistical power and thus may have been accountable for some of the negative findings described. In addition, the DS sample were characterised by having elevated levels of PTS, which might be representative of only a subgroup of patients with DS. In addition, the authors did not report the proportion of the DS group who also met criteria for a formal diagnosis of PTSD.

Of additional note is the fact that the DS group and the high-PTS control group both reported higher levels of general psychological distress compared to the low-PTS control group; however, these possible confounding factors were not incorporated into the analysis. Therefore, it remains possible that the between-groups differences could have reflected general psychological distress or another type of psychopathology (e.g. anxiety, depression). Moreover, whilst neurological disorders were excluded from the sample, it was unclear how many participants had other organic health problems. It also remains a possibility, therefore, that general medical conditions may have influenced the baseline RSA outputs. Finally, a potential design problem with Roberts et al's study was the presentation of random bursts of white noise throughout the experiment (detailed in a footnote in the published report). It is possible that these white noise bursts may have been distracting/unpleasant for the participants, and so may have influenced the results observed.

### **3.5.3. Summary: emotion in patients with dissociative seizures (literature review)**

When asked directly, patients with DS show heightened levels of a range of negative emotional states (e.g. depression, anxiety, fear, anger), in addition to considerable dysregulation of emotion. Moreover, symptoms of autonomic arousal and/or panic have also been found to be elevated in patients with DS during their seizures. In at least a proportion of patients with DS, identifying and describing emotions seems to be somewhat impaired (i.e. alexythmia).

Furthermore, experimental findings indicate an attentional bias towards facial emotion, in addition to behavioural avoidance of such stimuli. This bias may be linked to high levels of stress responsivity in this group (e.g. generally elevated HPA activation) and previous traumatic experiences. In addition, patients with DS experience elevated intensity in their emotional responses to some types of affective images (neutral/positive). As yet, there have been no functional neuroimaging studies of emotional processing in patients with DS, although this is an important area for future research.

### **3.6. An affective-dissociation model of dissociative seizures**

This section synthesises the currently available empirical and theoretical literature on the aetiology of DS. Table I outlines a proposed general explanatory framework incorporating the possible aetiological factors thought to contribute to the occurrence of DS. Within this framework, a variety of factors are proposed to increase the risk of developing DS (e.g. trauma, neurobiological factors, personality traits, psychopathology, dissociative tendencies, aberrant emotional processing – first column in Table I), although it is probable that these predisposing factors vary between patients. Furthermore, the various risk factors may interact in any one patient. For example, neurobiological factors may create a pre-existing vulnerability that combines with developmental trauma/stressors to cause a dissociative processing style, personality disorder and/or psychopathology. Moreover, the unique combination of these latter factors may be associated with abnormal emotional processing. It is proposed that aberrant processing of emotional stimuli may be a core predispositional factor for DS, by elevating overall levels of emotional distress and arousal.

The general predisposition towards psychological/somatoform dissociation could be shaped into seizure-like phenomena by symptom modelling and the iatrogenic influence of medical information (second column in Table I). Initial onset of the disorder might be precipitated by an acute increase in distress levels, which raises emotional distress to a ‘threshold’ level. The factors which might contribute to this elevated distress are shown in the third column of Table I. It is likely that in many cases, more than one of these factors might be precipitate DS onset. Table I also includes the variety of factors that might maintain/perpetuate the occurrence of seizures (fifth column) after the initial onset of the disorder. The framework presented in Table I also includes triggering factors for the occurrence of individual seizures (fourth column). The proposed triggering variables include acute states of stress/anxiety, aberrant emotional processing and crucially, dissociation of voluntary control and awareness of somatic/psychological processes. In order to further specify this possible triggering mechanism for individual DS episodes, Figure I illustrates a hypothetical affective-dissociation model of this process.

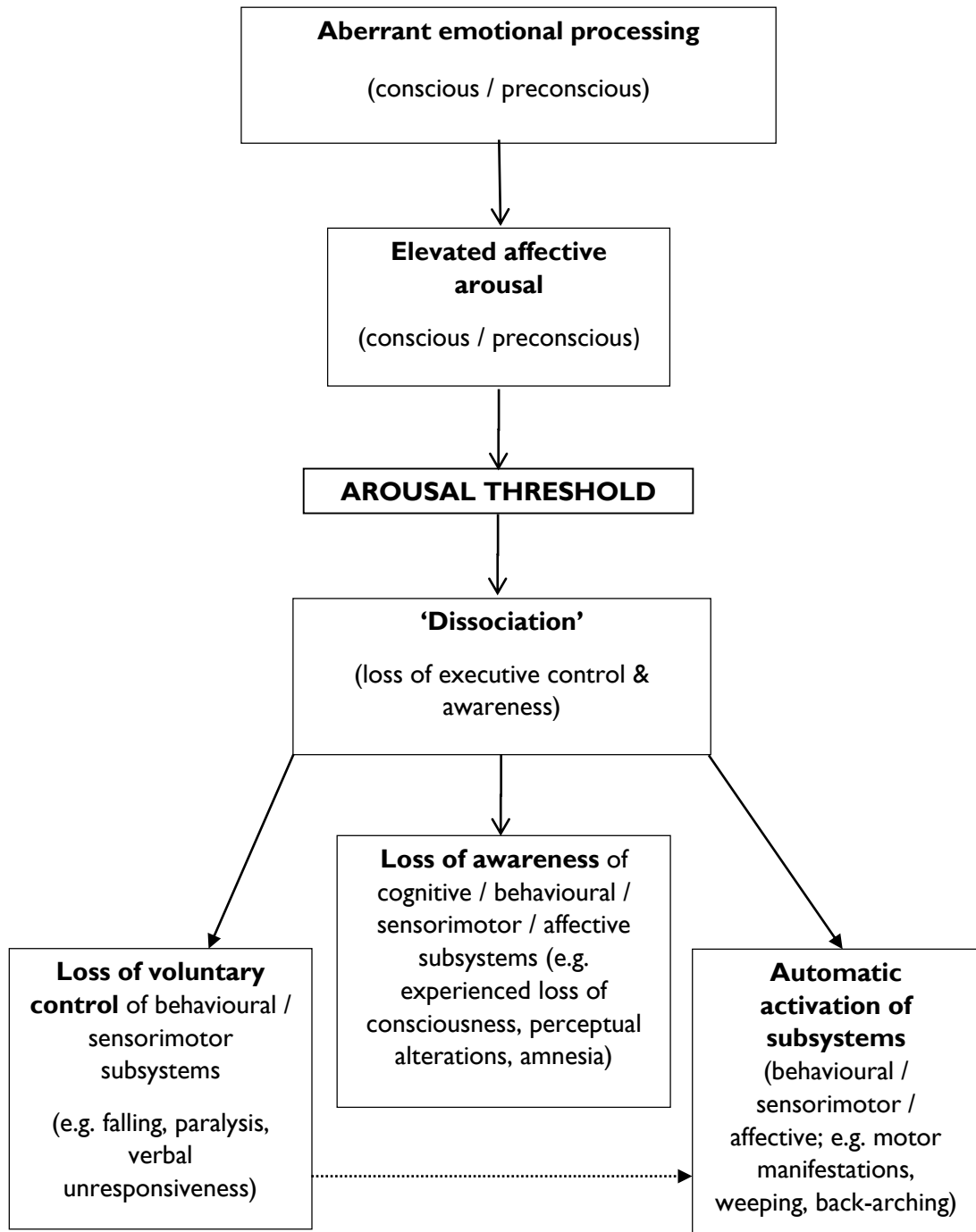
**Table 1. Aetiological factors in dissociative seizures**

| <b><i>Predisposing</i></b>                                                                                           | <b><i>Shaping</i></b>                                                              | <b><i>Precipitating</i></b>                                                                                      | <b><i>Triggering<sup>a</sup></i></b>                                      | <b><i>Maintaining*</i></b>                                                                   |
|----------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------|----------------------------------------------------------------------------------------------|
| Developmental trauma/stressors (e.g. abuse, neglect, family dysfunction, bereavement, medical trauma)                | Exposure to symptom models (e.g. familial ES, comorbid ES, other paroxysmal event) | Elevated distress levels (threshold)                                                                             | Aberrant emotional processing                                             | Primary and secondary gain (e.g. anxiety reduction, financial and social support, sick role) |
| Neurobiological factors (e.g. head injury, intellectual disability, genetics, cognitive impairment, substance abuse) | Iatrogenic influence of medical investigations and assessment (i.e. suggestion)    | Traumatic events or trauma-associated events                                                                     | Elevated affective arousal                                                | Ongoing elevated stress levels                                                               |
| Personality characteristics (e.g. avoidance, emotional dysregulation)                                                |                                                                                    | Medical or psychological crisis (e.g. life-threatening illness, severe pain, psychiatric diagnosis or admission) | Arousal threshold reached - dissociation of conscious awareness & control | Relationship dysfunction (e.g. enmeshment, conflict)                                         |
| Psychopathology (e.g. PTSD, anxiety, depression)                                                                     |                                                                                    | Head injury / neurological illness (e.g. epilepsy)                                                               |                                                                           | Maladaptive cognitions (e.g. locus of control, illness beliefs and attributions)             |
| Trait dissociation (somatoform / psychological)                                                                      |                                                                                    | Relationship crises / dysfunction                                                                                |                                                                           | Dysfunctional coping strategies (e.g. avoidance, emotion-focus)                              |
| Aberrant emotional processing                                                                                        |                                                                                    |                                                                                                                  |                                                                           | Psychopathology                                                                              |

<sup>a</sup>these triggering factors are inter-related

\*these factors are likely to be bidirectionally related to DS occurrence

**Figure 1. An affective-dissociation model of a DS episode**



At the top level of the model in Figure 1, it is proposed that aberrant emotional processing of environmental stimuli is present in patients with DS and is an important contributor to initiating individual seizures. One aspect of this might be preconscious hypervigilance for the detection of threat-related emotional signals from others (e.g. angry facial expressions), as suggested by Bakvis, Roelofs, et al., 2009. Furthermore, elevated physiological responding to affectively significant stimuli may also occur, but this may not be associated with subjective awareness of such responses (Goldstein and Mellers, 2006). A key hypothesis is that these bodily affective responses to significant stimuli, processed consciously or preconsciously, might result in an accumulation of affective arousal, which may or not be associated with subjective anxiety/distress in the patient. This accumulation of arousal might develop gradually (over hours or days) or rapidly (minutes), and may reach a hypothetical threshold level (Baslet, 2011). This threshold may represent the maximum level of emotional arousal/distress that is tolerable, and may vary from patient to patient. As patients may already experience heightened levels of arousal/distress in general, it is possible that relatively mild stressors or emotional stimuli could allow a rapid ascent towards this threshold.

Reaching the threshold of arousal is proposed to trigger the onset of a dissociative mechanism, constituting a temporary but severe loss of control and awareness over emotion, cognition, behaviour, and sensation. This represents the beginning of a DS episode. When affective arousal/distress reaches the threshold level, some patients may consciously allow the dissociative mechanism to be initiated (Stone & Carson, 2013); however, most patients will not experience any voluntary control over the initiation of this mechanism. The proposed dissociative mechanism would serve the psychological function of reducing conscious awareness of the intolerable emotional distress, but also gives rise to the dysfunctional by-products of reduced awareness and control of other behavioural, cognitive and bodily processes. Once the dissociative state has been initiated, it is likely that the patient will no longer have the ability to inhibit or terminate the ensuing symptoms.



In cognitive terms, when the threshold of arousal is reached, it may cause a direct disruption to the usual control processes taking place between higher and lower cognitive systems. This could constitute a temporary malfunction or inhibition of the 'executive control centre' (possibly lateral prefrontal cortex). At present, it is only possible to speculate how the elevated arousal levels might cause this disturbance. One possibility is via direct inhibitory outputs from limbic areas (e.g. amygdala) to the prefrontal cortex (PFC). On the other hand, it might be that the biochemical effects of heightened arousal (e.g. adrenalin and cortisol release) may have a disruptive influence on prefrontal functioning, at a given level. A similar hypothesis was suggested by Roelofs and Spinhoven (2007).

Regardless of the specific mechanism(s), this temporary 'shutting down' or malfunctioning of the executive control centre could explain the variety of symptom types observed in DS. On the one hand, loss of executive control over behavioural and sensorimotor subsystems could lead to losses in voluntary behaviour, such as falls, paralysis and verbal unresponsiveness (i.e. 'negative' behavioural symptoms of DS). In addition, the malfunctioning executive control system might automatically activate inappropriate learned behaviours (i.e. behavioural 'routines') or emotional responses, such as pelvic-thrusting, weeping, back-arching, screaming or 'tonic-clonic' type movements ('positive' DS symptoms). Loss of the usual top-down regulation/control that would typically modulate such behavioural and affective responses might permit these responses to continue autonomously during the DS, until executive control is once again restored (when the DS ends). During the seizure, losses of awareness of one or more subsystems (behavioural/sensorimotor/affective/cognitive) might underlie the amnesia, perceived losses of consciousness, and perceptual alterations reported by some patients (e.g. deafness, blindness; i.e. 'negative' cognitive symptoms of DS).

In terms of how this model relates to previous perspectives, in some respects, the current model attempts to integrate some of the basic ideas from cognitive models (e.g. R.J. Brown, 2002; Hilgard, 1994; Oakley, 1999), neurobiological perspectives (e.g. van der Kruis et al., 2012; 2014), and affective/dissociative models (e.g. Baslet, 2011; Goldstein and Mellers, 2006; Roberts and Reuber, 2014). As suggested by the cognitive

accounts, it is proposed that dysfunction in a superordinate control centre is crucial in the initiation of the dissociative response in DS. It is argued here that this dysfunction is the core underlying cause of the variety of phenomena observed during DS. However, a purely cognitive account, without reference to affective dysfunction is not an adequate explanation. Therefore, a core role for abnormal emotional responding (i.e. affective arousal) is proposed. As suggested by Baslet (2011) in relation to DS, the concept of a threshold of arousal is an important addition to the current model.

In the current thesis, DS are conceptualised as a transient dissociative state. The specific symptoms experienced by any one patient during such a state may vary, possibly depending on their unique neural organisation, connectivity patterns and learning history. For example, the losses of control and awareness might not affect all lower level subsystems in every seizure. The severity of an attack (i.e. the number, intensity and duration of symptoms experienced) may depend to some extent on the level of affective arousal present immediately prior to and during the DS. However, it is presently argued that DS cannot occur in the absence of dissociation, as proposed by Roberts and Reuber (2014), because DS are inherently a manifestation of dissociation (i.e. a psychologically-mediated loss of voluntary control and/or awareness).

### **3.7. Rationale, aims and hypotheses**

Many influential theorists now share the general idea that aberrant emotional processes, particularly related to autonomic arousal, are central to the aetiology of DS. However, as noted by other authors (e.g. Roberts et al., 2012), there is still a paucity of research into emotional processing in patients with DS. The overarching aim of the research presented in this thesis was, therefore, to further explore emotional processing in patients with DS, using experimental, laboratory-based techniques. The research sought to examine preconscious, subjective and autonomic emotional responses to affective stimuli, including facial expressions and more general affective images.

Investigating these processes was hoped to further inform the model proposed in Figure 1. Specifically, empirical evidence of altered affective processing in patients with DS would provide further support for the proposition that such alterations might serve to trigger dissociative responses in patients with DS, and thus, the hypothesis that such alterations represent the proximal cause of individual DS occurrence. Another important aim was to measure other relevant psychosocial characteristics of the group, in order to examine the validity of the general aetiological framework presented in Table 1. Moreover, the research sought to explore the extent to which these psychosocial variables related to any observed differences in emotional processing. Finally, an additional aim was to elicit patients' perceptions and interpretations of their emotional processing styles, and their understanding of how these processes relate to their symptoms, using qualitative techniques. This aim was included to ensure greater depth and a patient-centred perspective on the research topic. The previous empirical and theoretical literature, combined with the proposed model and explanatory framework outlined above, indicated several general hypotheses, as follows:

### ***Emotional responding/functioning***

1. Relative to healthy control participants, it was hypothesised that patients with DS would show an exaggerated preconscious attentional bias towards emotional facial expressions, particularly for threat-related expressions (i.e. anger). This hypothesis was suggested by previous research (e.g. Bakvis, Roelofs, et al., 2009).
2. Patients with DS were predicted to exhibit elevated autonomic responses to consciously processed emotional stimuli (i.e. skin conductance responses). Whilst this hypothesis was not supported by Roberts et al. (2012), the hypothesis required testing with additional psychophysiological methods (e.g. skin conductance responses), and in response to social stimuli (i.e. emotional facial expressions).

3. Patients with DS were hypothesised to show altered subjective emotional responses to consciously processed affective stimuli. This hypothesis was derived on the basis of previous studies (e.g. Prigatano & Kirlin, 2009; Roberts et al., 2012).

### ***Background aetiological factors***

4. Patients with DS were predicted to report higher levels of adverse life events (i.e. trauma, abuse, stressful experiences) and childhood family dysfunction compared to healthy controls. This would be expected on the basis of a large number of previous studies (see Chapter 2).
5. Patients with DS were expected to report higher levels of trait psychological and somatoform dissociation, compared to healthy controls (see section 3.3).
6. Patients with DS were predicted to exhibit higher levels of borderline personality tendencies and general psychological distress (anxiety/depression), compared to healthy control participants (see Chapter 2).

In order to examine hypotheses 1-3, three experimental laboratory-based studies were conducted. Hypothesis 1 was tested in an experiment measuring preconscious processing of emotional facial expressions (Chapter 6), which aimed to replicate and extend the findings of Bakvis, Roelofs, and colleagues (2009). This experiment utilised an emotional Stroop paradigm, and included a larger sample of patients and controls than is typically reported in the literature ( $n = 40$  in each group). Furthermore, a second experimental study (Chapter 7) assessed autonomic and subjective responses to consciously processed facial expressions of emotion, in order to test hypotheses 2 and 3. A third experiment (Chapter 8) examined autonomic and subjective responses to consciously processed affective images, in order to further examine hypotheses 2 and 3, with more varied stimuli. Together, these studies would provide findings of relevance to the top level depicted in Figure 1 (i.e. proposed aberrant emotional processing).

Hypotheses 4-6 were examined with the use of self-report measures. The findings relating to these psychosocial variables are summarised in Chapter 5. The use of these measures had the potential to confirm and extend some of the factors proposed in the aetiological framework presented in Table I. Moreover, a qualitative study of patients' perceptions and understanding of the role of emotions in DS is presented in Chapter 9. This study aimed to explore patients' insights into the possible emotional mechanisms and aetiological factors associated with DS, and also to generate novel hypotheses for testing in future studies.

The present research also sought to explore whether there were relationships between emotional responding (as measured experimentally) and the psychosocial variables measured with self-report questionnaires. Additional exploratory aspects of the research included an examination of potential relationships between aspects of emotional responding, psychosocial factors and seizure-related variables (e.g. seizure frequency, ictal symptoms, total seizure symptoms). These exploratory analyses are reported in Chapters 5-8.

## **Chapter 4. General methodology**

### **4.1. Pilot study**

Following confirmation of ethical approval (see section 4.2.1), a small pilot study was conducted including six control participants and two patients diagnosed with DS. The aims of the pilot study were to assess the practical and technical feasibility of the tests and procedures, and to ensure the acceptability of the measures from participants' perspectives. On completion of piloting with control participants, some modifications were made to the procedures, in order to reduce the overall amount of time taken for testing, and to make the tasks more acceptable for participants. Where modifications to the procedures were made, these are detailed in the methodology sections of the relevant chapters. The summary given below is based on the final procedures used throughout the research.

### **4.2. Participants**

#### **4.2.1. Recruitment process and ethical approval**

The quantitative aspects of the research were approved by the local research ethics committee (Joint South London and Maudsley & Institute of Psychiatry Research Ethics Committee; reference 08/H0807/82) in December 2008. Recruitment for the quantitative studies commenced in January 2009 and continued until December 2012. A break in recruitment occurred from May 2010 to January 2011 due to the research student taking maternity leave. The qualitative study received approval from the same committee in June 2010 as a substantial amendment to the original application. Recruitment of patients for the qualitative study commenced in January 2011 and continued until December 2012. Approval letters for the quantitative and qualitative aspects of the research can be found in Appendices 1 and 2.

Before taking part, all participants received an information sheet, usually one to two weeks prior to being contacted again. Potential participants were reassured that participation was voluntary and that there was no obligation to agree. For those who wished to participate, written informed consent was obtained, after thoroughly

discussing the study with the research student and having the opportunity to ask questions. Appendices 3 to 8 include information sheets and consent forms for the quantitative and qualitative studies.

On completion of the research, participants were reimbursed for time and loss of earnings at a rate of £30 for the quantitative studies (all completed in one testing session) or £15 for the qualitative study (completed during a separate session). Reimbursement was provided by the Department of Psychology (Institute of Psychiatry) as part of the research student's postgraduate studentship. An award was also obtained from the Central Research Fund (University of London) in June 2009 for additional funding of travel expenses. Therefore, as of July 2009, all participants were offered up to £20 for travel expenses, when receipts were provided. This modification was approved as a substantial amendment by the local ethics committee in July 2009, and the approval letter can be found in Appendix 9. The additional funding of travel expenses facilitated recruitment of the clinical sample, particularly because many patients travelled to London from considerable distances for clinical and research appointments.

Patients diagnosed with DS were recruited from two clinical services in south London: the Department of Neuropsychiatry (outpatients) at the Maudsley Hospital, and the Department of Psychological Medicine at King's College Hospital. Both services were part of the South London and Maudsley NHS Foundation Trust. Consultants, registrars and junior doctors were made aware of the nature of the research and the inclusion/exclusion criteria, and identified potentially suitable candidates during assessment appointments or shortly thereafter.

Initially, clinicians provided information sheets to patients during clinical appointments, by post, and/or requested permission for the research student to contact them about participation. The research student then contacted patients within three weeks (but no less than one week) by telephone, unless the patient requested otherwise. During the initial telephone conversation, a summary of the research procedures was provided, along with an overview of the possible disadvantages and advantages of participation. At this stage, patients were able to ask

any questions or raise concerns, and the voluntary nature of the research was reiterated. It was then determined whether the patient was willing to participate.

For those who agreed, an appointment was made for the quantitative session. Written informed consent was obtained before participants commenced the research protocol. Participants consenting to participate completed all quantitative aspects of the research in one session (reported in chapters 5-8). This approach was taken in order that the required sample size of patients ( $n = 40$ ) could be included, to ensure adequate statistical power for the study, in addition to the time constraints of data collection within a doctoral research programme.

On completion of the first (quantitative) research session, patients were asked if they would consider taking part in the second, qualitative session (once recruitment for the latter study had commenced). Patients who indicated an interest were then provided with the information sheet for the qualitative study (see Appendix 7) and the research student then contacted them between one and three weeks later to confirm whether they wished to participate. If so, the same procedure took place as for quantitative research session. Only a subsample of patients completed the qualitative study.

Recruitment of healthy control participants was carried out via advertisement (see Appendix 10 for the wording of the advertisement), either on fliers distributed in the local area, or using adverts placed on local sections of internet websites such as Gumtree ([www.gumtree.com](http://www.gumtree.com)). The Institute of Psychiatry volunteer database (Mindsearch; [www.mindsearch.iop.kcl.ac.uk](http://www.mindsearch.iop.kcl.ac.uk); discontinued service) was also used for recruitment of some participants. Potential candidates were initially screened for the eligibility criteria (see below) either by telephone or email. This included several consistent screening questions, requesting self-disclosure of relevant details. Potential candidates were assured of the confidentiality of this information.

Individuals not fully meeting the study eligibility criteria were thanked for their interest and provided with a brief explanation as to why they were not being invited to participate. For those who met the study criteria, an information sheet and



consent form was provided and the research student contacted them between one and three weeks later to discuss it further. For those who agreed to take part, an appointment was made and a confirmation letter sent.

#### **4.2.2. Eligibility criteria**

The following criteria were used to determine the eligibility of patients for this study:

- having received a diagnosis of DS based on video-EEG monitoring or on the basis of consensus clinical opinion of two consultant neuropsychiatrists / neurologists
- aged between 18-65 years
- estimated intelligence quotient (IQ) scores of 70 or greater
- fluency in English
- absence of documented history of / current epilepsy or other major neurological conditions (e.g. degenerative diseases, significant traumatic brain injury)
- absence of significant medical illness that may have affected the measures used (e.g. hyperthyroidism)
- absence of current major depression, anxiety, substance dependence, or psychosis (these diagnoses are known to be associated with differences in emotional or cognitive functioning which may have confounded performance on the experimental measures)
- having not undergone psychological treatment for DS at the time of recruitment

The criteria applied for the determination of eligibility of control participants were as follows:

- between 18-65 years of age
- IQ scores of 70 or greater
- absence of self-reported current / previous psychiatric diagnoses or treatment
- absence of confirmed history of / current epilepsy or other neurological conditions
- absence of current (self-reported) major medical conditions (e.g. cardiovascular disease, hyperthyroidism)

All participants had normal or corrected-to-normal vision. Control participants were matched to the patient sample as well as possible for the following demographic details: age, gender, handedness, socioeconomic status (using the simplified National Statistics – Socioeconomic Classification; NS-SEC; Office for National Statistics, 2010), smoking status (yes/no) and years of education. Matching of demographic characteristics was important due to the potential influence of these variables on the psychosocial variables and affective processing tasks. For example, affective responsivity has been found to be influenced by gender (Kret & De Gelder, 2012; Stevens & Hamann, 2012; Whittle et al., 2011), and age (Nashiro, Sakaki, & Mather, 2011; Silvers et al., 2012; Sullivan, Ruffman, & Hutton, 2007). Cigarette smoking was recorded for all participants due to its possible effects on ANS functioning (e.g. Kotamäki, 1995; Niedermaier et al., 1993).

#### **4.3. Materials and measures**

At the start of the first research session, a questionnaire was administered verbally to all participants, with the purpose of obtaining a detailed description of the sample, including those variables on which participants were to be matched (e.g. age, gender, education). The following information was gathered in the questionnaire:

1. full name and date of birth
2. current age (years)
3. gender (male, female, other)
4. ethnic background: white, mixed ethnic backgrounds, Asian/Asian British, black/African/Caribbean/black British, other
5. Handedness (self-report: right, left, ambidextrous)
6. Marital status (self-report: single, married, separated, cohabiting, widowed)
7. Years of full-time education (or part-time equivalent)
8. Highest qualification attained
9. Current or most recent occupation (or that of the highest household wage earner)
10. Medical conditions (current)

11. Prescribed medication (current)
12. Psychiatric diagnoses (current, previous)
13. Neurological illness/injury (current, previous)
14. Smoking status (yes/no)

For the patient sample, verification for some of the above (e.g. items 11, 12, 13 and 14) and additional relevant clinical details were obtained by inspecting the medical case notes (after consent was obtained). The following additional details were also recorded:

- Age at seizure onset
- Current seizure frequency (per month)
- Diagnostic investigations undergone (i.e. routine-EEG, ictal-EEG, video-EEG, structural neuroimaging such as computed tomography or MRI)

The study described in Chapter 5 involved the administration of several self-report questionnaires, which were completed by participants and scored manually by the research student. All three experimental tasks (chapters 6-8) were conducted using an Acer laptop with a 17 inch screen. These experiments were programmed with E-Prime experimental software (Psychology Software Tools, Incorporated). Experimental stimuli were prepared using Microsoft Paint or Adobe Photoshop software, where necessary.

Participants' subjective or behavioural responses during testing were recorded with the integral laptop keyboard (Chapters 7 and 8) or with voice key registration (Chapter 6). The psychophysiological measure (skin conductance responses; Chapters 7 and 8) was obtained with a Powerlab data acquisition system and associated LabChart data analysis software (ADInstruments). Quantitative data were initially exported into Excel software (Microsoft Office) for initial organisation and extraction. Subsequently, all statistical analyses of the quantitative data were conducted using the Statistical Package for the Social Sciences, versions 20-22 (SPSS; IBM). Qualitative data were collected with a digital recording device (Sony ICD-

P520), transcribed verbatim into Microsoft Word files, and then analysed using paper-and-pen methods and NVIVO (10<sup>th</sup> edition) software (QSR International, Limited).

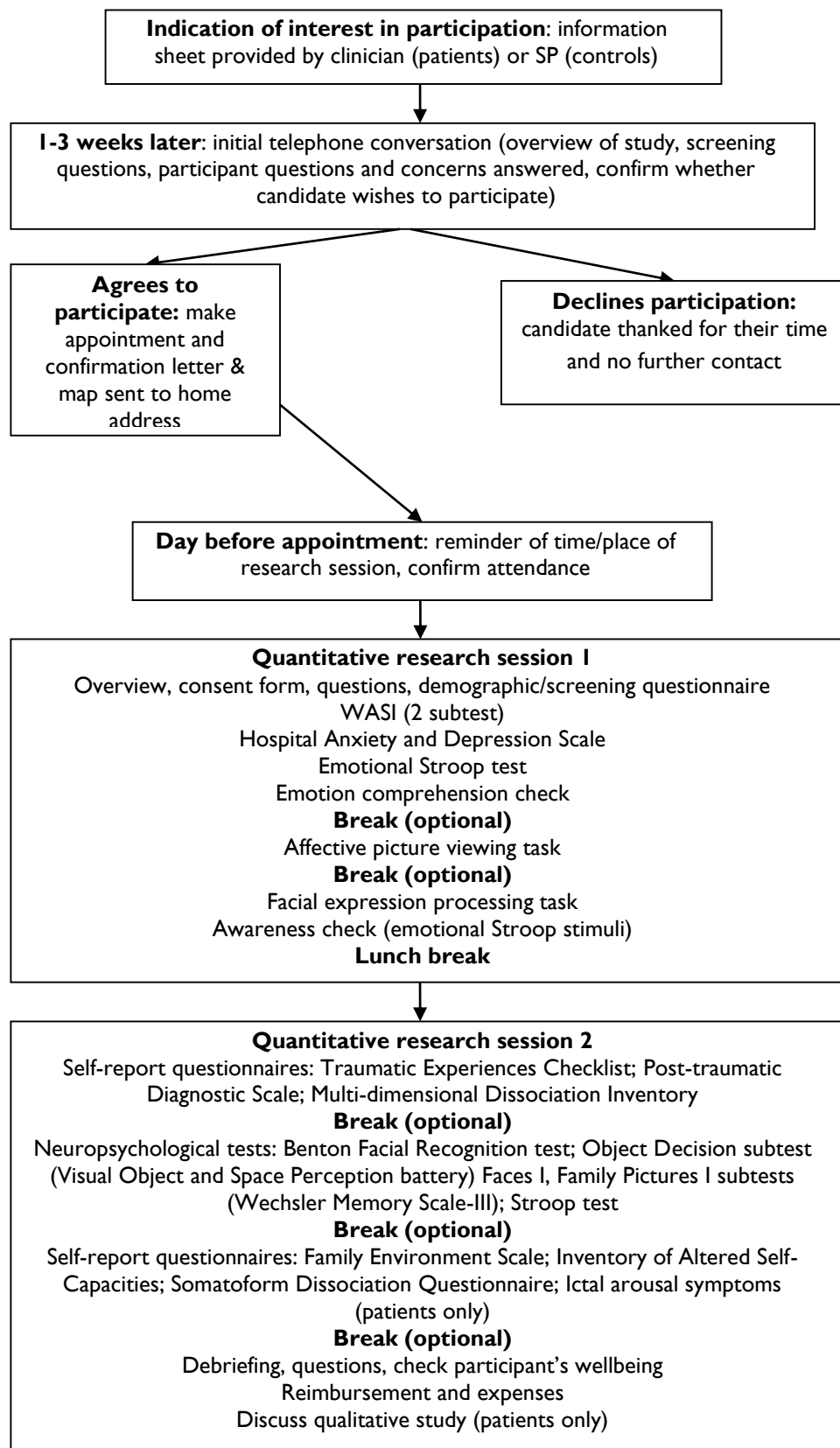
#### **4.4. Research design and procedure**

The self-report questionnaire (Chapter 5) and experimental studies (Chapters 6-8) shared the same overall research design. These studies adopted a between-groups cross-sectional approach. All four studies included just one between-groups factor with two levels (diagnostic status: patient, control). The within-subjects factors differed between studies and are detailed in the relevant chapters, as are the dependent variables. The qualitative study (Chapter 9) involved recruitment of a subgroup of patients with DS, and the data were collected using semi-structured interviews.

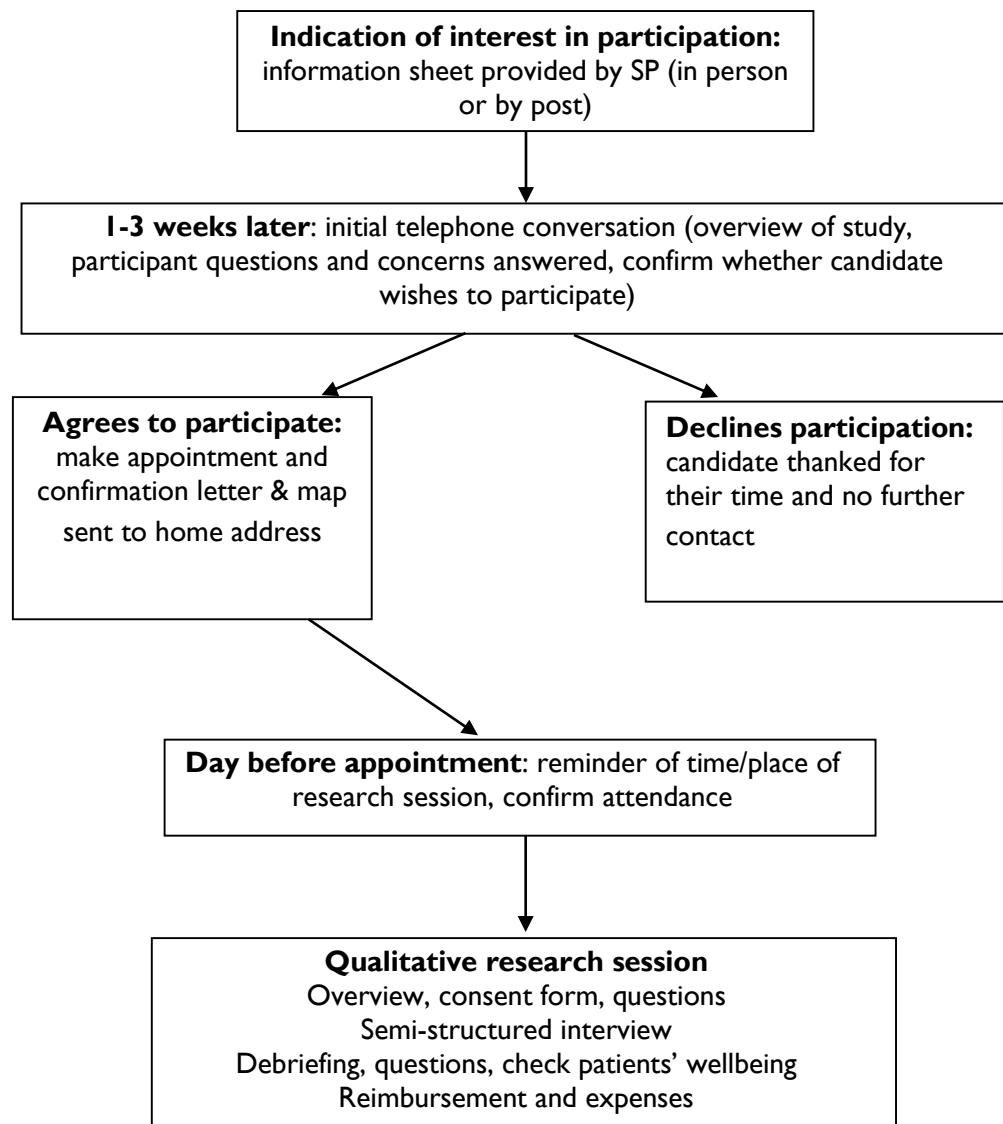
The overall procedures for the quantitative and qualitative research sessions are detailed in Figures 2 and 3 respectively. Within the quantitative research session, the order of administration of tests was devised with consideration of the following issues: the requirement of obtaining psychophysiological measurements at approximately the same time of day and in uniform environmental conditions for every participant, potential order and interference effects, and ethical reasons. All participants were requested to avoid smoking, taking caffeine or eating large meals within the hour prior to attending the research setting for the quantitative tests, due to the potential effects on the psychophysiological data.

Participants were offered several breaks during the quantitative session, due to its considerable length. Furthermore, the research student monitored the wellbeing of participants throughout both research sessions. Participants were reminded that if they wished to withdraw at any time, not complete a given test, or return on another occasion to complete the research, they were welcome to do so without giving a reason. Any participants showing signs of fatigue or distress were always given the opportunity to take a break or discontinue testing. A list of local and national support and advice services was also offered, if participants indicated any sign of distress. All participants were thanked for their time and reimbursed the specified amount.

**Figure 2. Flowchart of the overall procedure for the first (quantitative) research session**



**Figure 3. Flowchart of the overall procedure for the second (qualitative) research session (clinical sample only)**



## **4.5. Data analysis**

### **4.5.1. General analytical techniques**

Between-group comparisons on demographic and psychosocial variables were conducted with independent samples t-tests, chi-square, or Mann-Whitney tests. Furthermore, analyses of experimental data (Chapters 6-8) were carried out with mixed factorial Analysis of Variance (ANOVA) or Analysis of Covariance (ANCOVA), with group (DS, control) as the between-groups factor and with different within-subjects variables for each experiment. Where interactions or main effects were significant in the overall analysis, the simple effects were examined using one-way ANOVA or multiple pairwise comparisons (e.g. t-test). In these main analyses, an alpha level of  $p < .05$  was set as the criterion for significance, with p-values between .05 and .06 reported as non-significant trends. When multiple tests were carried out, the family-wise error rate was taken into account by either adjusting the p-values with Bonferroni-Hochberg corrections (Hochberg, 1988) or adopting a more stringent alpha level when assessing significance (i.e.  $p < .01$ ). Trends were not considered in such analyses.

Exploratory correlational analyses adopted Pearson's or Spearman's correlations, depending on whether the scores were normally distributed. Moreover, Chapter 5 included several exploratory univariate and multivariate logistic regression analyses, with the outcome variable representing group status (DS, control) and the predictor variables being scores on the self-report measures described in that chapter. Some chapters report exploratory univariate and multivariate linear regression analyses. Again, an alpha level of  $p < .05$  was adopted for determination of significance in the multivariate analyses (trends defined by p-values of .05 - .06); however, when multiple univariate analyses were carried out, a more stringent alpha level was used ( $p < .01$ ) to control for familywise error.

Qualitative data were analysed using techniques based on Interpretative Phenomenological Analysis (IPA; Smith & Osborn, 2008). Further detail about this approach can be found in Chapter 9.

#### **4.5.2. Power calculations**

Regarding the self-report data presented in Chapter 5, the power calculation indicated that an independent samples t-test would have 80% power to detect a large effect size (0.8), at an alpha level of  $p < .01$ , with a sample size of 39 per group (GPower 3.1; Faul et al., 2007; 2009). When DS patients have previously been compared to healthy controls on the DES (Bernstein & Putnam, 1986), for example, a large effect size of approximately 0.81 was demonstrated with only 20 participants in each group (Goldstein et al, 2000). Therefore, the inclusion of 40 participants in each group in the self-report analyses would seem to provide adequate power to detect large effect sizes, should they be present.

The between-groups analysis strategy (i.e. ANOVA, ANCOVA) utilised in Chapters 6-8 was based on the following power calculation: for a two group ANCOVA, at  $p=0.05$ , a total sample of 74 was estimated to have 80% power to detect a medium effect size of 0.5 (G-Power 3.1; Faul et al., 2007; 2009). For the dependent measures included in the current research, the detection of such an effect size was considered sufficient. Unfortunately, it was not possible to estimate an effect size for the results of Bakvis, Roelofs, et al. (2009) because the exact mean and standard deviation values were not provided in the report (although they were presented graphically). Moreover, there are as yet no published studies of explicit facial expression processing in patients with DS. However, Sierra et al. (2006) found effect sizes of approximately 3.64 and 9.33 for intensity ratings and SCRs respectively, in responses to disgusted facial expressions in patients with depersonalisation disorder relative to healthy controls (effect sizes calculated using Cohen's  $d$ , with the control standard deviation as the denominator).

Regarding affective picture viewing tests, on the basis of the descriptive data provided by Roberts et al. (2012), an estimated effect size of 1.08 was calculated for intensity ratings for neutral images (patients with DS relative to healthy controls). Moreover, an approximation of the effect size obtained by Sierra and colleagues (2002) for SCR responses to unpleasant affective (IAPS) stimuli (depersonalisation disorder versus controls, 15 participants in each group) was 2.71. For the same stimuli, the effect size for participants' subjective arousal ratings was approximately 1.18. Therefore, it



would seem that if a between-group effect is present, large effect sizes are likely to be observed, and the analytic strategy described above (ANOVA, ANCOVA) would be adequately powered to detect such differences with the proposed sample size.

With regards to power considerations for the exploratory correlational analyses, according to GPower (v3.1; Faul et al., 2007; 2009), a sample size of 27 is sufficient to detect a correlation of  $r = 0.6$  at  $p < .01$  with 80% power. As 26 was the lowest sample size included in most exploratory correlational analyses, it can be concluded that these analyses were sufficiently powered to detect relationships between variables, should those relationships exist. However, some of the exploratory analyses may have been somewhat under-powered due to the unexpected but necessary analysis of psychophysiological data in subgroups of participants (Chapters 7 and 8).

For logistic regression analyses (Chapter 5), a commonly accepted standard is the inclusion of a minimum of 10 events per variable (EPV; Hosmer & Lemeshow, 2000; Peduzzi et al., 1996). The EPV refers to the frequency of the least common outcome (i.e. in this instance the value is 40, representing the number of DS patients included), divided by the number of predictor variables. Therefore, with 40 ‘events’ (i.e. cases of DS), the inclusion of only four predictors could be justified in the multivariate logistic regression analyses. Whilst some authors have suggested that lower EPV values may be sufficient to obtain stable and accurate regression coefficients (Vittinghoff & McCulloch, 2006), the 10 EPV rule was adopted to avoid any risk of ‘overfitting’ the models obtained. Moreover, according to Field (2013; based on GPower output), multivariate linear regression analyses with a sample size of 40 are adequately powered to detect a large effect size ( $R^2 = .26$ ), with four predictors. Therefore, once again, the multivariate regression analyses presented in Chapters 6-8 included a maximum of four predictor variables in each model.

#### **4.5.3. Data exploration and assumption checks**

Initial exploration of all quantitative data involved visual inspection for input errors or outliers using boxplots and/or histograms. Any clear data input errors were corrected. Outlying data points (defined by z scores of 3.29 or more; Tabachnick &

Fidell, 2007) were removed from the relevant dataset if this significantly improved the distribution of the data. If the distribution was not significantly improved by removing the data points, the outlying values were retained. In cases where outliers were retained, the relevant analyses were carried out twice, once including and once excluding the outlying data points. If no clear differences in results emerged, the results of the full dataset were reported. If there was a difference in results, the findings from the modified dataset (i.e. outliers removed) were reported.

For continuous numerical dependent variables, the normality assumption was assessed visually (histograms, Q-Q plots) and statistically using the Shapiro-Wilk (S-W) test. The S-W test was chosen because it has been found to be the most powerful of the four most readily available tests (Razali, Nornadiah, & Wah, 2011). If the S-W test was significant (i.e.  $p < .05$ ), the distribution was assumed to be non-normal. Skewness or kurtosis z-scores over 1.96 also indicated significant deviation from normality and facilitated identification of the nature of the violation (Field, 2013). Furthermore, where appropriate, the assumption of homogeneity of variances (homoscedasticity) was assessed with Levene's test. If the test statistic was significant ( $p < .05$ ), it was assumed that heteroscedasticity was present and that this assumption was not met.

If a non-normal distribution was present, or if there was significant heteroscedasticity, transformations were carried out to attempt to correct these features. The transformations used were Naperian Logarithm, Logarithm (10), square root, square, cube and reciprocal. If the transformed scores resolved the violations, these transformed scores were used in the analyses. However, if the transformations did not improve the data, then untransformed scores were analysed with non-parametric techniques (i.e. Mann-Whitney U) if appropriate. Any transformations conducted are detailed in the relevant results sections. In cases where the use of non-parametric techniques was not appropriate (e.g. analysis of the factorial experimental data, mixed factorial ANCOVA), the planned parametric tests were carried out. The use of ANOVA or ANCOVA with data that deviate somewhat from normality can be justified by the finding that ANOVA/ANCOVA models are generally robust to such violations (e.g. Glass, Peckham, & Sanders, 1972; Levy, 1980; Schmider et al., 2010).

Moreover, Central Limit Theorem indicates that when sample sizes are sufficiently large (degrees of freedom of  $>20$ ), one can assume the normality of the sampling distribution despite the appearance of non-normality in the distribution of the raw data (Tabachnick & Fidell, 2007).

Where relevant (repeated measures/within-subjects variables), the assumption of sphericity was tested using Mauchly's test. If this assumption was violated (i.e. a significant result on Mauchly's test), corrections were made using the most appropriate technique. Following Field (2013), the Greenhouse-Geiser correction was used if the estimate of sphericity was well under 0.75, whereas the Huynh & Feldt technique was applied where this estimate was equal to or exceeded 0.75.

When regressions were carried out, the variables were checked for multicollinearity (Field, 2003; D. Howell, 1997). Relevant diagnostics (tolerance, variance inflation factor (VIF) were checked to ensure that they were within acceptable limits ( $VIF < 10$ ; tolerance  $> 0.2$ ), according to Myers (1990) and Menard (1995) respectively, as cited by Field (2013). After completing multivariate regression analyses, several additional assumptions were checked. Cook's distances were checked for all cases, to confirm the absence of influential cases on the regression statistics. Cook's distance of greater than 1 was used as the criterion for influential cases (Tabachnik & Fidell, 2007).

## **Chapter 5. Psychosocial factors in patients diagnosed with dissociative seizures: a comparison with healthy individuals**

### **5.1. Introduction**

#### **5.1.1. Background**

As discussed in Chapters 2 and 3, a number of methodological weaknesses and inconsistencies have been noted in the existing literature on psychosocial factors in patients with DS. In relation to trauma history, some studies have reported exclusively on one or few specific types of life event (i.e. abuse and neglect), often failing to assess other potentially important experiences (Akyuz et al., 2004; Driver-Dunckley et al., 2011; Gazzola et al., 2012; Koby et al., 2010; Stone et al., 2004). Other studies have reported on a wider range of life events, but have not provided explicit criteria on which traumatic events are defined (e.g. Bora et al., 2011; LaFrance & Syc, 2009; Turner et al., 2011). Additionally, many studies have not included data on the perceived impact or significance of reported life events, although some investigators have examined symptoms of PTSD (e.g. Dikel et al., 2003; Rosenberg et al., 2000).

In previous research, validated measures have not been used consistently to measure trauma, with retrospective analysis of case notes frequently reported (e.g. An et al., 2010; Driver-Dunckley et al., 2011; Gazzola et al., 2012; Krahm et al., 1997; Lancman et al., 1993; O'Sullivan et al., 2007; Salinsky et al., 2012; Scheepers et al., 1994; Thomas et al., 2013). Lack of matching for gender has also been an important weakness in some instances (e.g. Alper et al., 1993; Dixit et al., 2013; Gazzola et al., 2012; Kaplan et al., 2013; Litwin & Cardena, 2000). Moreover, many investigators have not assessed the extent to which trauma/abuse may have been associated with clinical characteristics such as DS severity. Finally, analysis of whether the relationship between trauma and DS was mediated by other relevant variables (i.e. somatisation, general psychological distress) was often not included in previous work.

A variety of methods have also been used to examine family functioning in patients with DS. Whilst standardised self-report measures have been most common (e.g.

LaFrance et al., 2011; Salmon et al., 2003), qualitative techniques (e.g. J.L. Griffith et al., 1998; Stanhope et al., 2003) and interviews (e.g. Bowman & Markand, 1999) have also been described. In some instances, specification as to whether the measures referred to childhood, current or lifetime family functioning has not been included (e.g. Hovorka et al., 2007; LaFrance et al., 2011). Furthermore, some authors have not examined family functioning in relation to other psychosocial characteristics of the patient group, such as psychopathology, personality characteristics or trauma history (e.g. Krawetz et al., 2001). However, one particularly interesting study suggested that family dysfunction (i.e. elevated control) may mediate the relationship of abuse to DS (Salmon et al., 2003).

When examining psychopathology and personality characteristics in patients with DS, some previous investigators have not included control groups (e.g. Alessi & Valente, 2013; Bowman & Markand, 1999; Ettinger, Dhoon, et al., 1999; Lempert & Schmidt, 1990; Snyder et al., 1994). Once again, a wide range of techniques have been used to assess these characteristics, including standardised or unstandardised interviews (e.g. Abubakr et al., 2003; Baillés et al., 2004; Dworetzky et al., 2005), medical case note review (e.g. Alessi & Valente, 2013; Dixit et al., 2013; Seneviratne et al., 2011), and self-report measures (e.g. Hixson et al., 2006; LaFrance & Syc, 2009; Prueter et al., 2002).

### **5.1.2. Rationale, aims and hypotheses**

One objective of the current study was to further explore adverse life experiences in patients with DS, by using a standardised measure with sound psychometric properties (Traumatic Experiences Checklist; TEC; Nijenhuis et al., 1999). The measure assesses the presence of a wide range of life events; however, since not all events on the measure would be considered universally traumatic (e.g. poverty, divorce), the term 'adverse' life events will be used when discussing the findings on this measure. An advantage of this questionnaire is that it assesses the perceived impact of each reported life experience, and thus provides an insight into subjective responses to life events. In the current study, gender was also carefully matched between groups, to control for the known association between gender and trauma/abuse history. Furthermore, symptoms of PTSD were assessed with a

standardised self-report measure (Post-traumatic Diagnostic Scale; PDS; Foa et al., 1997), in order to further explore the psychological impact of such events.

Additionally, following Salmon et al. (2003), subscales from a psychometrically-sound measure of family functioning (Family Environment Scale; FES; Moos & Moos, 1981) were included in the study, which allowed clear hypotheses to be generated regarding the aspects of dysfunction that were likely to be observed (e.g. expressiveness, control). A measure of psychological dissociation that had not previously been used with DS patients was also administered (Multiscale Dissociation Inventory; MDI; Briere, 2002), in order to assess several dimensions of psychological dissociation in this group. Furthermore, a measure of somatoform dissociation was also included (SDQ-20; Nijenhuis et al., 1996), so that somatic manifestations of dissociation were captured.

In order to explore personality functioning, a measure was selected that assesses aspects of behaviour/personality commonly associated with borderline personality disorder (Inventory of Altered Self-Capacities; IASC; Briere, 2000). This measure was included to gain insight into which specific aspects of borderline 'type' tendencies may be particularly important in patients with DS. Finally, a measure of the specific symptoms experienced by patients during their seizures was also included (Goldstein & Mellers, 2006), in order to examine whether any of the psychosocial variables were related to specific manifestations of the disorder.

An important overall aim of the study was to assess these variables in patients with DS relative to healthy individuals. On the basis of previous research findings, the following hypotheses were tested:

### **I. Adverse life experiences:**

- i. Patients diagnosed with DS were expected to report significantly more lifetime adverse life experiences than healthy control participants.
- ii. Rates of sexual, physical and emotional abuse were predicted to be significantly higher in the DS group, relative to healthy controls.

## **2. Childhood family functioning:**

Elevated reports of childhood family control and reduced childhood family expressiveness were predicted in the DS group, relative to healthy controls.

## **3. Psychological dissociation:**

The DS group were predicted to report significantly higher levels of psychological dissociation than the healthy control group, as measured by the MDI.

## **4. Somatoform dissociation:**

A significant elevation in somatoform dissociation was predicted in the DS group, relative to the healthy control group.

## **5. Psychological distress:**

Symptoms of anxiety and depression were hypothesised to be higher in the DS sample, compared to healthy controls.

## **6. Borderline personality tendencies:**

The DS group were predicted to exhibit elevations in borderline personality traits, as measured by the IASC, compared to healthy controls. On the basis of previous literature, affect dysregulation was expected to be higher in the patients than in the control group.

In addition, the research sought to explore the ways in which these psychosocial variables related to each other in the DS group. Moreover, a further objective was to carry out an exploratory assessment of the relative importance of these variables in differentiating patients with DS from healthy controls. Therefore, those subscales/scales showing significant between-group differences were examined further in exploratory analyses. Correlations were used to examine whether there were inter-relationships between the psychosocial variables, and whether any were associated with seizure-related factors (i.e. seizure frequency, length of seizure disorder, seizure symptoms). Moreover, exploratory logistic regression analyses

were used to assess which variables best differentiated patients with DS from healthy controls, after accounting for the influence of other possibly confounding variables (e.g. years of education).

## **5.2. Methodology**

### **5.2.1. Participants**

Details regarding participant recruitment and ethical approval can be found in Chapter 4 (section 4.2.1).

### **5.2.2. Measures**

All measures included in the study were self-report questionnaires, as described below:

#### ***Traumatic Experiences Checklist (TEC; Nijenhuis et al., 1999)***

This questionnaire provides an assessment of 29 types of potentially traumatic experiences, and has good psychometric properties. The internal consistency is satisfactory (Cronbach's  $\alpha = 0.86-0.9$ ) and good concurrent validity is evident, for example, total TEC scores correlated strongly with the Stressful Life Events Screening Questionnaire (Goodman et al., 1998;  $r = 0.77$ ). From this measure, it was possible to identify the total number of traumatic experiences participants recalled (0-29) and also to examine the self-reported impact of those experiences (1-5; none-extreme).

#### ***Post-traumatic Diagnostic Scale (PDS; Foa et al., 1997)***

The PDS measures the presence and/or severity of current symptoms of Post-traumatic Stress Disorder (PTSD), within the previous month. The measure provides an overall PTSD symptom score, but also incorporates criteria used to determine a diagnosis of PTSD according to the Diagnostic and Statistical Manual for Mental Disorders – fourth edition (DSM-IV; American Psychiatric Association, 1994). Participants who described moderate-extreme impact of at least one life event on the TEC were asked to complete the PDS.



The authors report good internal consistency for total scores (0.92), re-experiencing (0.78), avoidance (0.84) and arousal (0.84) subscales. The three subscales correlate significantly with each other and with total symptom scores ( $r = 0.73-0.94$ ;  $p < .001$ ). Test-retest coefficients were found to be satisfactory (Kappa = 0.77-0.85). The PDS has acceptable sensitivity (0.89) and specificity (0.75).

### ***Family Environment Scale (FES; Moos & Moos, 1981)***

The full FES comprises 10 subscales, assessing internal family processes, and how these relate to the external social environment. There are three forms of the questionnaire; including a 'real' form (how the family is perceived to be in reality), an 'ideal' form (how the individual would like the family to be), and an 'expected' form (how the family is expected to be in new situations). The real form of this questionnaire was used in this study. Patients were asked to complete the questionnaire with reference to their childhood family context.

Several subscales were selected, based on previous research (Salmon et al., 2003), including:

- i. Cohesion: the extent to which family members act as a cohesive unit.
- ii. Expressiveness: the level of expression of emotions, thoughts and feelings.
- iii. Conflict: the presence of overt expressions of anger or hostility within the family.
- iv. Independence: the degree of autonomy and assertiveness in family members.
- v. Control: the use of rules and regulatory strategies in the family context.

Each subscale includes nine statements for which respondents must provide a dichotomous response (true/false). These answers yield scores between 0-9, which can then be converted to T-scores. The measure assesses perceived family functioning only, from the perspective of the participant.

### ***Hospital Anxiety and Depression Scale (HADS; Zigmond and Snaith, 1983)***

This scale was developed for use in general medical settings, measuring current (non-somatic) symptoms of anxiety and depression. It consists of 14 items, with seven

items examining anxiety and seven measuring depression. Participants rate the extent to which they have experienced each symptom in the previous week. Both subscales have a maximum score of 21, with scores of 8-10 identifying borderline/doubtful cases, and scores of 11-21 indicative of definite caseness. The subscales show good internal consistency (Cronbach's alpha: anxiety = 0.78-.093; depression = 0.76-0.9 (Mykletun et al., 2001; Zigmond & Snaith, 1983). It was predicted that the DS sample would report greater levels of general somatic complaints; therefore, this test was selected in order to avoid the anxiety and depression scores being inflated by physical symptoms.

### ***Multiscale Dissociation Inventory (MDI; Briere, 2002)***

This 30-item scale measures a variety of psychological dissociative symptoms. Participants rate the extent to which they have experienced each symptom during the last month. Each of the six subscales has a maximum score of 25 and a minimum of 5. The raw scores are converted to T-scores. All six of the following subscales were administered:

- i. Disengagement: cognitive and/or emotional detachment from the immediate situation and stimuli.
- ii. Depersonalisation: Feeling separated from or alien to one's own body or self.
- iii. Derealisation: feeling as though the environment and the stimuli within it are unreal or dream-like.
- iv. Emotional Constriction: a marked reduction in awareness and experience of emotions (positive or negative).
- v. Memory Disturbance: experiencing memory lapses (without organic cause).
- vi. Identity Dissociation: unstable identity states, experiencing more than one 'self'.

For subscales 1-5 above, T-scores above 80 are considered clinically significant. For subscale 6 (Identity Dissociation), a T-score above 95 suggests clinical relevance. The psychometric properties of the scale suggest that the subscales are reliable across several samples (Cronbach's alpha ranging from .77-.92). The MDI subscales also correlated positively with scores on several other dissociation measures, including

the DES ( $r = .66-.81$ ), and the Trauma Symptom Inventory – Dissociation subscale (TSI DIS; Briere et al., 1995;  $r = .62-.76$ ), suggestive of good convergent validity.

### ***Somatoform Dissociation Questionnaire (SDQ-20; Nijenhuis et al., 1996)***

This scale measures the presence of physical symptoms which are conceptualised as resulting from somatoform dissociation. These symptoms are those which would typically be considered medically unexplained. Respondents rate the frequency of such symptoms in the previous year. The SDQ-20 provides a score ranging from 20-100 (20 items each with scores ranging from 1-5). Higher scores are indicative of elevated somatoform dissociation. Cronbach's alpha for this scale was reported as .95, indicative of good internal consistency. Scores from the SDQ-20 were also reported to correlate positively with scores on the Dissociation Questionnaire (Nijenhuis et al., 1996), suggesting adequate convergent validity.

### ***Inventory of Altered Self-Capacities (IASC; Briere, 2000)***

This scale assesses abilities that are involved with difficulties in the maintenance/regulation of identity, emotions, relationships and behaviour. The IASC questionnaire provides scores on seven subscales which can be categorised into three groups, as follows:

#### **I. Relatedness**

- i. Interpersonal Conflict: Reports of problems in relationships (e.g. the tendency to have turbulent, emotionally distressing relationships).
- ii. Idealisation-Disillusionment: Ambivalent feelings towards significant others (e.g. oscillating from extremely positive to extremely negative responses).
- iii. Abandonment Concerns: Sensitivity to abandonment by others (real, perceived); anticipation or anxiety about the loss of close interpersonal relationships.

#### **2. Identity**

- i. Identity Impairment: Difficulties in maintaining a stable experience of personal identity and self, across time and situations.
- ii. Susceptibility to Influence: Tendencies towards being guided or unduly influenced by others, without sufficient consideration of the self.

### 3. Affect control

- i. Affect Dysregulation: Deficits in control and regulation of affect (e.g. intense mood swings, expression of anger, problems terminating unpleasant affective states).
- ii. Tension reduction activities: Externalisation of emotional distress (e.g. binge-eating, sexual or self-injurious behaviours).

Each IASC subscale comprises nine items, which are scored from 1-5. Respondents indicate the frequency of each symptom in the previous six months. Therefore, the total score for the scale is between 9 and 45. These scores are converted to T-scores. The psychometric properties of the IASC include Cronbach's alpha values ranging from .78-.93 for the seven subscales, suggesting good internal consistency (Briere & Runtz, 2002). The average Cronbach's alpha values were .89, .93, and .89 in normative, clinical and university samples respectively. Scores on all of the IASC subscales correlated significantly with the 'borderline features' subscale of the Personality Assessment Inventory (Morey, 1991) with values ranging from .61 to .86 (all significant at  $p < .01$ ).

#### ***Ictal symptoms (Goldstein & Mellers, 2006)***

Seizure symptoms were assessed in the DS group using an abbreviated form of the questionnaire developed by Goldstein & Mellers (2006; see Appendix II). The questionnaire measures various symptom types, including: autonomic arousal, symptoms relating to the chest/abdomen, aspects of mental state, cognitive phenomena, and general seizure symptoms. Each type of symptom is assessed with respect to patients' most recent and most severe attacks. The authors' psychometric details on this scale indicated Cronbach's alpha ranging from .621 to .883 across the subscales, suggestive of moderate to good reliability.

#### **5.2.3. Design and procedure**

The overall research design, procedures and order of administration of the questionnaires was described in Chapter 4 (section 4.4). The dependent variables within this study were the scores obtained on the scales/subscales described above.

#### **5.2.4. Data analyses**

The between-group data analyses, including statistical techniques, were also described in Chapter 4 (section 4.5). Variables that differed significantly between-groups were considered for examination in the exploratory analyses. The exploratory correlational analyses utilised non-parametric (Spearman's rho) bivariate correlations, to assess inter-relationships between significant psychosocial variables (TEC, PDS, HADS, MDI, SDQ-20, IASC), and seizure-related variables (seizure frequency, duration of disorder, ictal symptoms). Logistic regression analyses were conducted with group as the outcome variable (DS, control). Variables which had been significant in the between-groups analyses were first tested with univariate binary logistic regression analyses, with YoE controlled by entry in the first step. Variables that were significant in the univariate analyses ( $p < .01$ ), and/or had theoretical significance, were then entered as predictors in multivariate binary logistic regressions. Predictors were entered in blocks, with input of variables within blocks using the forced entry procedure.

Mediation analyses (Field, 2013) were carried out for some variables. The PROCESS custom dialog box (Hayes, 2012) for SPSS was installed and used to obtain the relevant statistics. Where mediation effects were investigated, Sobel's test (1982, as cited by Field, 2013) determined the significance of the effects.

### **5.3. Results**

#### **5.3.1. Demographic characteristics**

A summary of participants' demographic characteristics can be found in Table 2. No significant difference in age was found between the DS and control groups. Furthermore, the ratio of males to females did not differ between groups. The proportion of participants with right-hand dominance also did not differ significantly between groups, neither did the proportion of smokers. The ethnic backgrounds of the two groups can be found in Figure 4. There was no significant difference in the proportion of participants describing themselves as 'white' between-groups.

**Table 2. Demographic characteristics of the total sample (by group)**

|                               | <b>DS (n = 40)</b>                                               | <b>Control (n = 43)</b>                                             | <b>Test statistics</b>              |
|-------------------------------|------------------------------------------------------------------|---------------------------------------------------------------------|-------------------------------------|
| Age (years)                   |                                                                  |                                                                     |                                     |
| Mean (SD)                     | 38.1 (13.1)                                                      | 36.9 (11.9)                                                         | U (83) =                            |
| Median (IQR)                  | 40 (23)                                                          | 36 (20)                                                             | 806, p = .622                       |
| Gender                        | Male = 8 (20%)<br>Female = 32 (80%)                              | Male = 8 (18.6%)<br>Female = 35 (81.4%)                             | $\chi^2$ (1, n=83) = .026, p = .872 |
| Handedness                    | Right = 30 (75%)<br>Left / ambidextrous = 10 (25%)               | Right = 38 (88.4%)<br>Left / ambidextrous = 5 (11.6%)               | $\chi^2$ (1, n=83) = 2.5, p = .114  |
| Ethnicity                     | White = 32 (80%)<br>Non-white = 8 (20%)                          | White = 28 (65.1%)<br>Non-white = 15 (34.9%)                        | $\chi^2$ (1, n=83) = 2.29, p = .130 |
| YoE                           |                                                                  |                                                                     |                                     |
| Mean (SD)                     | 12.9 (2.5)                                                       | 14 (2.6)                                                            | U (83) =                            |
| Median (IQR)                  | 12.5 (3)                                                         | 14 (5)                                                              | 631, p = .035                       |
| Qualifications                | GCSEs / none = 16 (40%)<br>Further / higher = 24 (60%)           | GCSEs / none = 9 (20.9%)<br>Further / higher = 34 (79.1%)           | $\chi^2$ (1, n=83) = 3.58, p = .058 |
| Marital status                | Currently single = 24 (60%)<br>Long-term relationship = 16 (40%) | Currently single = 34 (79.1%)<br>Long-term relationship = 9 (20.9%) | $\chi^2$ (1, n=83) = 3.58, p = .058 |
| Socio-economic status (NSSEC) | 1 = 18 (45%)<br>2,3,4 or 5 = 22 (55%)                            | 1 = 18 (41.9%)<br>2,3,4 or 5 = 25 (58.1%)                           | $\chi^2$ (1, n=83) = .083, p = .773 |
| Smoking                       | Yes = 11 (27.5%)<br>No = 29 (72.5%)                              | Yes = 9 (20.9%)<br>No = 34 (79.1%)                                  | $\chi^2$ (1, n=83) = .489, p = .484 |

SD = standard deviation

IQR = interquartile range

YoE: years of full-time education (or equivalent)

DS = dissociative seizures

NSSEC: National Statistics Socio-economic Classification system

1 = Higher managerial, administrative and professional occupations

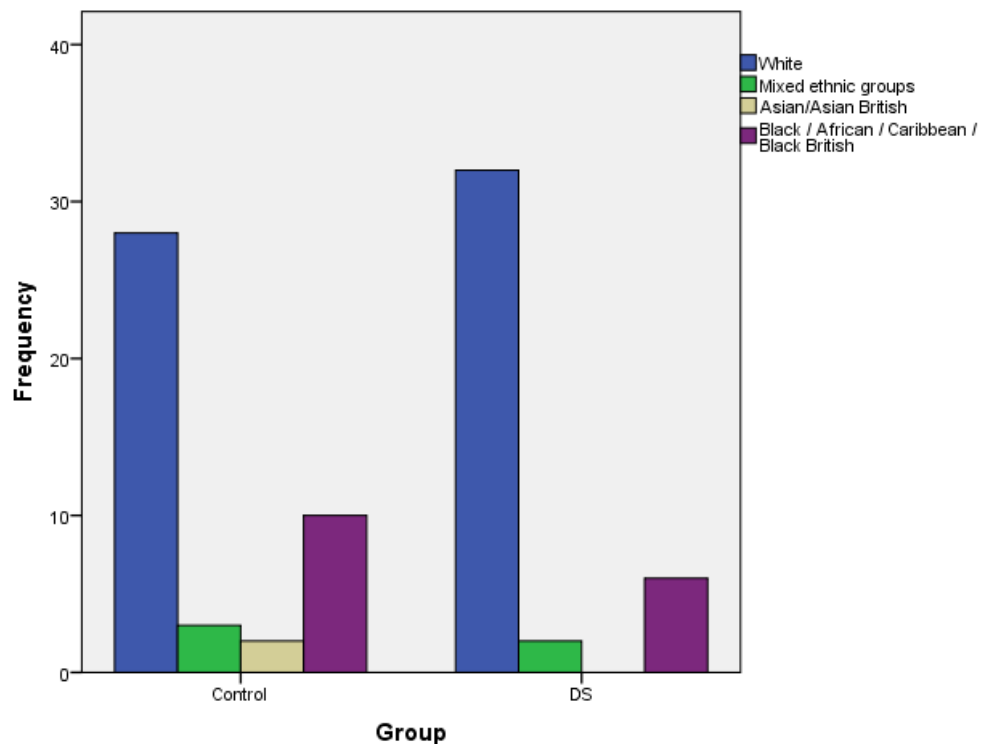
2 = Intermediate occupations

3 = Small employers and own account workers

4 = Lower supervisory and technical occupations

5 = Semi-routine and routine occupations

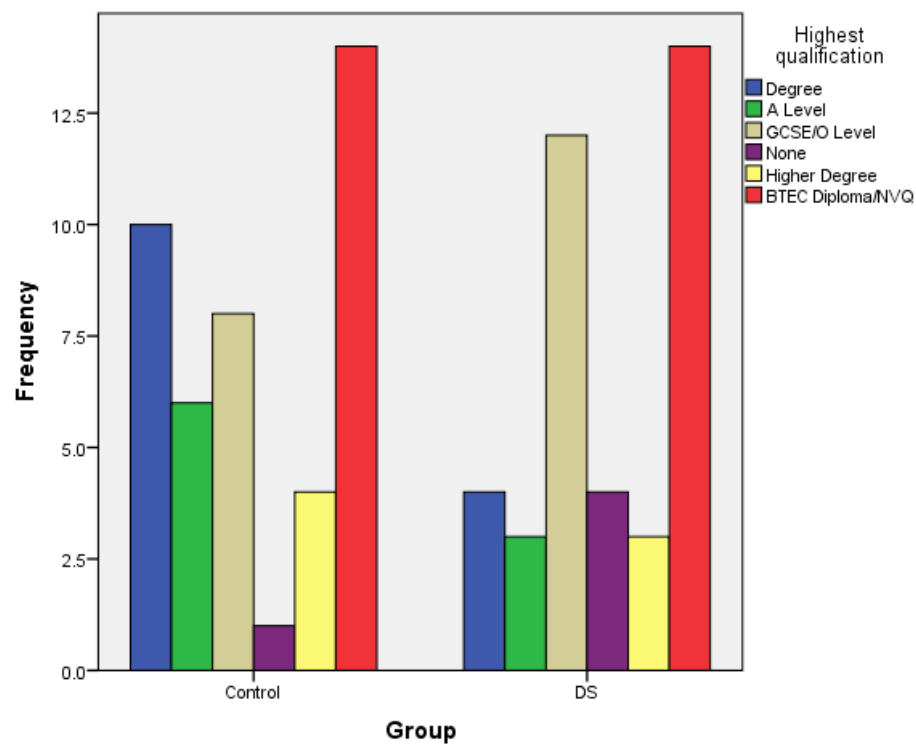
**Figure 4. Self-reported ethnicity by group**



The DS group reported significantly fewer years of full-time education (YoE) than control participants. Figure 5 shows educational details for the two groups. A non-significant trend indicated that attainment of further/higher education was somewhat more common in the control group, relative to the DS group. In addition, a non-significant trend indicated that patients with DS were more likely to be in a current long-term relationship (married/cohabiting), relative to the control group. Details regarding the marital status of participants are available in Figure 6.

On the basis of self-reported occupation, socioeconomic status (SES) was classified using the five class version of the National Statistics - Socioeconomic Classification system (NS-SEC; Office for National Statistics, 2010). Participants were classified on the basis of their current/most recent occupation. Those who were financially dependent on/interdependent with another party (e.g. spouse or parents), were classified according to the occupation of the highest wage earner in the household. Those who were long-term unemployed or had never worked due to medical reasons (including DS) were placed into category 5.

**Figure 5. Highest educational qualification by group**



**Figure 6. Marital status by group**

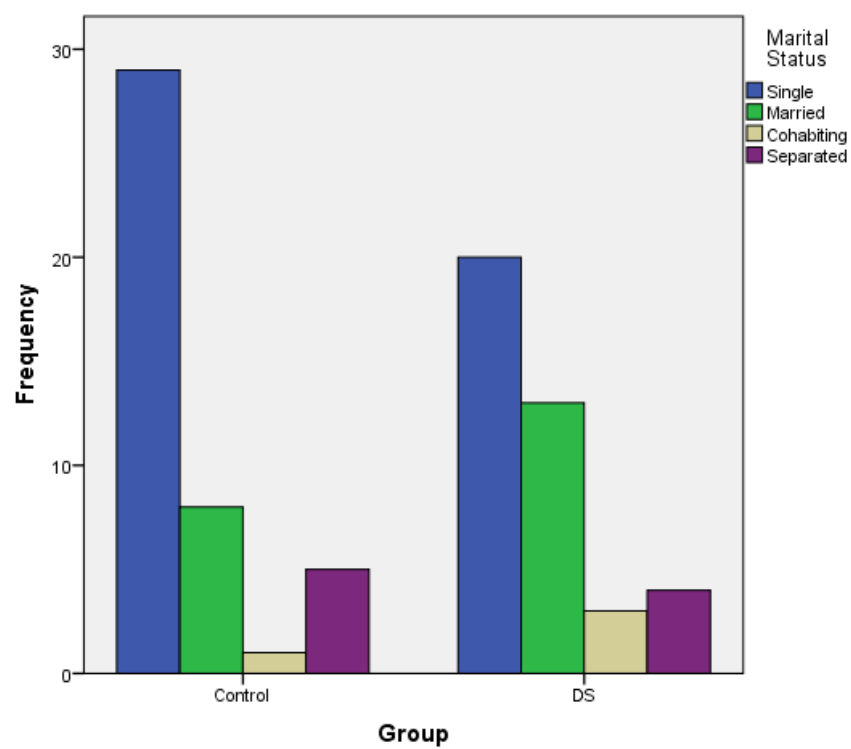
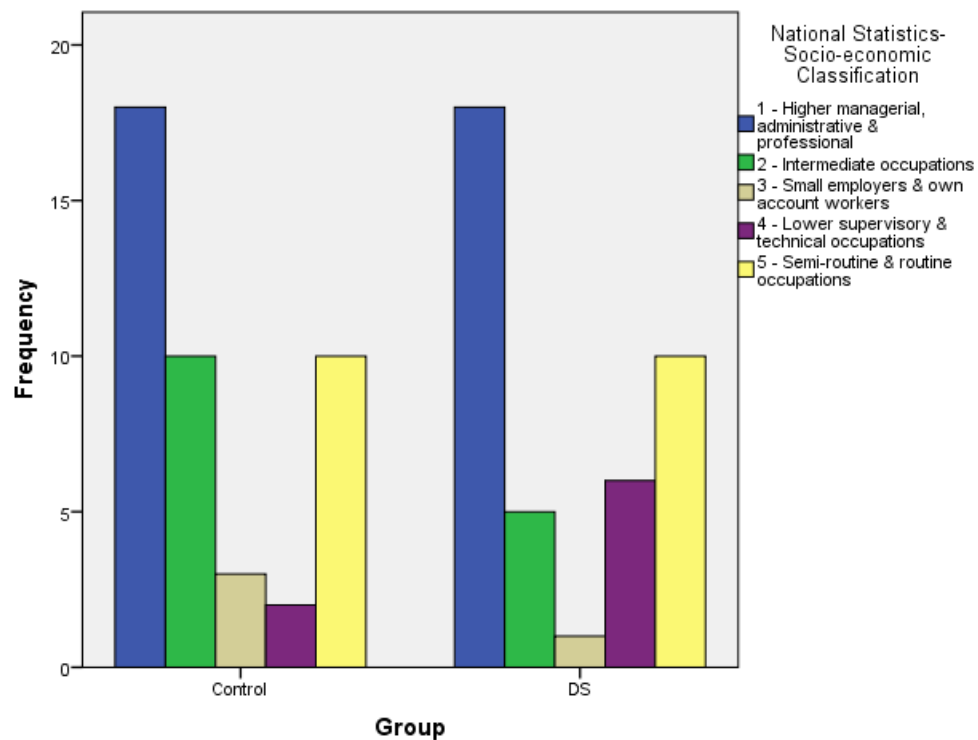




Figure 7 displays the number of participants (by group) in each of the five categories included in the NS-SEC. The proportion of participants belonging to category 1 did not differ significantly between groups.

**Figure 7. Socioeconomic status by group**



### 5.3.2. Clinical details

#### ***Medication and medical diagnoses***

A significantly larger proportion of DS patients were currently taking prescribed medication, relative to controls. Details on medication use can be found in Table 3. A significantly greater percentage of participants in the DS group reported a current medical diagnosis, relative to controls. The medical diagnoses disclosed by participants in each group are detailed in Table 4.

#### ***Seizure-related characteristics (DS group)***

The mean (SD) current seizure frequency reported by the patient sample (per month) and the total duration of the seizure disorder (in months) can be found in Table 5. The number of patients who underwent specific diagnostic tests for DS are also presented in the same table.

**Table 3. Medication details by group**

|                       | <b>Dissociative seizures<br/>(n = 40)</b>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     | <b>Control<br/>(n = 43)</b>                                                                                                                                                                                | <b>Test<br/>statistics</b>                   |
|-----------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------|
| Medication            | Yes = 29 (72.5%)<br>No = 11 (27.5%)                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           | Yes = 10 (23.3%)<br>No = 33 (76.7%)                                                                                                                                                                        | $\chi^2$ (1,<br>n=83) =<br>20.2, p <<br>.001 |
| Medication<br>details | Anti-epileptic = 17<br>(42.5%)<br>Anti-depressant = 16<br>(40%)<br>Proton pump inhibitor =<br>8 (20%)<br>Analgesics and anti-<br>inflammatories = 8 (18%)<br>Hypertension reduction<br>= 5 (12.5%)<br>Benzodiazepine = 4<br>(10%)<br>Asthma medication = 4<br>(10%)<br>Oral contraceptive = 4<br>(10%)<br>Anti-dopaminergic = 2<br>(5%)<br>Cholesterol reducers = 2<br>(5%)<br>Hormonal treatment = 2<br>(5%)<br>Anti-psychotic = 1 (2.5%)<br>Anti-allergy = 1 (2.5%)<br>Anti-spasmodic = 1<br>(2.5%)<br>Diuretic = 1 (2.5%)<br>Anti-vertigo = 1 (2.5%)<br>Anti-cholinergic = 1<br>(2.5%)<br>Adrenergic agonist = 1<br>(2.5%) | Asthma medication = 4<br>(9.3%)<br>Anti-allergy = 1 (2.3%)<br>Hormonal treatment = 1<br>(2.3%)<br>Anti-retroviral = 1 (2.3%)<br>Cholesterol reducers = 1<br>(2.3%)<br>Hypertension reduction =<br>1 (2.3%) |                                              |

**Table 4. Medical diagnoses by group**

|                           | <b>Dissociative seizures<br/>(n = 40)</b>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   | <b>Control<br/>(n = 43)</b>                                                                                                                      | <b>Test<br/>statistics</b>          |
|---------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------|
| Medical diagnoses present | Yes = 23 (57.5%)<br>No = 17 (42.5%)                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         | Yes = 6 (14%)<br>No = 37 (86%)                                                                                                                   | $\chi^2 (1, n=83) = 17.3, p < .001$ |
| Medical diagnoses details | Gastrointestinal = 6 (15%)<br>Hypertension = 5 (12.5%)<br>Previous hysterectomy = 5 (12.5%)<br>Asthma = 4 (10%)<br>Polycystic ovary syndrome = 2 (5%)<br>High cholesterol = 2 (5%)<br>Previous ovarian cancer = 2 (5%)<br>Alopecia = 1 (2.5%)<br>Hernia = 1 (2.5%)<br>Arthritis = 1 (2.5%)<br>Hypothyroidism = 1 (2.5%)<br>Erythema nodosum = 1 (2.5%)<br>Dyslexia = 1 (2.5%)<br>Allergies = 1 (2.5%)<br>Trigeminal nerve damage = 1 (2.5%)<br>Brugada Syndrome = 1 (2.5%)<br>Keratoconus = 1 (2.5%)<br>Amenorrhea = 1 (2.5%)<br>Hemochromatosis = 1 (2.5%) | Asthma = 4 (9.3%)<br>Osteoporosis = 1 (2.3%)<br>HIV = 1 (2.3%)<br>Hypertension = 1 (2.3%)<br>High cholesterol = 1 (2.3%)<br>Allergies = 1 (2.3%) |                                     |

**Table 5. Details of seizure disorder (dissociative seizures group)**

| <i>Current seizure frequency / month</i>                                                                                                                   |                             |
|------------------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------|
| Mean (SD)                                                                                                                                                  | 19.5 (47)                   |
| Median (IQR)                                                                                                                                               | 4.2 (14)                    |
| <i>Duration of seizure disorder (months)</i>                                                                                                               |                             |
| Mean (SD)                                                                                                                                                  | 89.3 (86.6)                 |
| Median (IQR)                                                                                                                                               | 54 (90)                     |
| <i>Diagnostic tests</i>                                                                                                                                    |                             |
|                                                                                                                                                            | Video-EEG = 27 (67.5%)      |
|                                                                                                                                                            | Imaging (MRI/CT) = 32 (80%) |
|                                                                                                                                                            | Routine EEG = 36 (90%)      |
| SD = standard deviation; IQR = interquartile range<br>video-EEG: video-electroencephalography CT = computed tomography<br>MRI = magnetic resonance imaging |                             |

### 5.3.3. Psychosocial characteristics: between-group comparisons

#### *Traumatic Experiences Checklist (TEC)*

The statistics for the TEC are shown in Table 6. The DS group reported significantly more total adverse life experiences and higher mean impact scores, relative to the control group. In addition, significantly more participants reported having experienced sexual and physical abuse in the DS group, in comparison to controls. However, there were no significant between-group differences in emotional neglect and abuse.

#### *Post-traumatic Diagnostic Scale (PDS)*

Statistical information for the PDS is available in Table 7. There was a significant elevation of Total, Re-experiencing, Avoidance, and Arousal PTSD symptoms in the DS group, relative to healthy controls. In addition, none of the control participants met the criteria for a diagnosis of PTSD, whilst 66.7% of the DS patients completing the measure did.

#### *Family Environment Scale (FES)*

The statistics for the FES data are shown in Table 8. After controlling for multiple comparisons, no between-groups differences were observed on any subscale.

**Table 6. Traumatic Experiences Checklist (TEC) scores**

|                          | <b>DS</b>   | <b>Control</b> | <b>Test statistics</b>                  |
|--------------------------|-------------|----------------|-----------------------------------------|
|                          | n = 39      | n = 43         |                                         |
| <b>Total</b>             |             |                |                                         |
| Mean (SD)                | 8.33 (4.67) | 5.69 (3.92)    | t (80) = -2.12, p =                     |
| Median (IQR)             | 8 (6)       | 5 (5.25)       | .037                                    |
| <b>Sexual abuse</b>      |             |                |                                         |
| n (%)                    | 20 (52.6%)  | 7 (16.3%)      | $\chi^2(1, n = 81) = 12,$<br>p = .001   |
| <b>Physical abuse</b>    |             |                |                                         |
| n (%)                    | 24 (61.5%)  | 14 (32.6%)     | $\chi^2(1, n=82) = 6.91,$<br>p = .009   |
| <b>Emotional abuse</b>   |             |                |                                         |
| n (%)                    | 26 (66.7%)  | 26 (60.5%)     | $\chi^2(1, n = 82) =$<br>.339, p = .560 |
| <b>Emotional neglect</b> |             |                |                                         |
| n (%)                    | 25 (64.1%)  | 19 (44.2%)     | $\chi^2(1, n = 82) =$<br>3.26, p = .071 |
| <b>Impact ratings</b>    |             |                |                                         |
| Mean (SD)                | 4.04 (0.7)  | 3.4 (0.9)      | U (83) = 493, p =                       |
| Median (IQR)             | 4.2 (1.1)   | 3.6 (1)        | .002                                    |

SD = standard deviation; IQR = interquartile range; DS = dissociative seizures

### ***Hospital Anxiety and Depression Scale (HADS)***

Table 9 displays the statistical details for the HADS analyses. There were highly significant elevations in Anxiety and Depression scores in the DS group relative to healthy control participants; however, the mean scores in the DS group were only in the borderline range for the Anxiety subscale and in the average range for Depression.

**Table 7. Post-traumatic Diagnostic Scale (PDS) scores**

|                        | <b>DS</b><br>n = 29 | <b>Control</b><br>n = 28 | <b>Test statistics</b> |
|------------------------|---------------------|--------------------------|------------------------|
| <b>Total</b>           |                     |                          |                        |
| Mean (SD)              | 23.6 (10.1)         | 7.5 (6.9)                | t (55) = -7.17, p <    |
| Median (IQR)           | 24 (11.5)           | 5 (9.8)                  | .001                   |
| <b>Re-experiencing</b> |                     |                          |                        |
| Mean (SD)              | 6.3 (4.5)           | 2.5 (2.8)                | U (57) = 190.5, p <    |
| Median (IQR)           | 6 (7.5)             | 2 (2)                    | .001                   |
| <b>Avoidance</b>       |                     |                          |                        |
| Mean (SD)              | 9.1 (5.02)          | 2.4 (2.6)                | U (57) = 87.5, p <     |
| Median (IQR)           | 8 (7)               | 1.5 (4.7)                | .001                   |
| <b>Arousal</b>         |                     |                          |                        |
| Mean (SD)              | 8.2 (3.5)           | 2.5 (3.8)                | U (57) = 107.5, p <    |
| Median (IQR)           | 9 (5)               | 1 (4.5)                  | .001                   |

SD = standard deviation; IQR = interquartile range; DS = dissociative seizures

### ***Multiscale Dissociation Inventory (MDI)***

Statistics for the MDI scores can be found in Table 10. There were significant differences on all subscales, with the DS group consistently reporting higher scores than control participants. The average scores were in the clinically significant range for the Disengagement, Depersonalisation, and Memory Disturbance subscales in the DS group, whereas the average scores were in the normal range for all subscales in the control group.

### ***Somatoform Dissociation Questionnaire (SDQ-20)***

Table 10 also displays statistics for the SDQ-20. When all SDQ-20 items were included in the analysis, there was a significant elevation in scores in the DS group relative to the control group. Furthermore, when the seizure-related item was removed, the significant difference remained ( $p < .001$ ).

**Table 8. Family Environment Scale (FES) scores**

|                     | <b>DS</b><br>n = 36 | <b>Control</b><br>n = 43 | <b>Test statistics</b><br>unadjusted (adjusted<br>where different) |
|---------------------|---------------------|--------------------------|--------------------------------------------------------------------|
| <b>Cohesion</b>     |                     |                          |                                                                    |
| Mean (SD)           | 36.8 (17.7)         | 43.5 (18)                | U (79) = 598.5, p =                                                |
| Median (IQR)        | 38 (34)             | 45 (34)                  | .082 (p = .328)                                                    |
| <b>Expression</b>   |                     |                          |                                                                    |
| Mean (SD)           | 39.9 (15.9)         | 41.7 (16.3)              | U (79) = 713.5, p =                                                |
| Median (IQR)        | 40 (29.5)           | 40 (25)                  | .549 (p = .575)                                                    |
| <b>Conflict</b>     |                     |                          |                                                                    |
| Mean (SD)           | 56.9 (15.2)         | 49.7 (11.1)              | U (79) = 554.5, p =                                                |
| Median (IQR)        | 57 (26)             | 49 (21)                  | .03 (p = .15)                                                      |
| <b>Independence</b> |                     |                          |                                                                    |
| Mean (SD)           | 41.4 (15.9)         | 44.1 (15.5)              | U (79) = 705, p = .491                                             |
| Median (IQR)        | 45 (24)             | 45 (16)                  | (.575)                                                             |
| <b>Control</b>      |                     |                          |                                                                    |
| Mean (SD)           | 54.7 (14.9)         | 52.9 (14.5)              | U (79) = 717.5, p =                                                |
| Median (IQR)        | 51.5 (22)           | 54 (22)                  | .575                                                               |

SD = standard deviation; IQR = interquartile range; DS = dissociative seizures

**Table 9. Hospital Anxiety and Depression Scale (HADS) scores**

|                   | <b>DS</b><br>n = 40 | <b>Control</b><br>n = 43 | <b>Test statistics</b> |
|-------------------|---------------------|--------------------------|------------------------|
| <b>Depression</b> |                     |                          |                        |
| Mean (SD)         | 7.02 (4.2)          | 2.3 (2.4)                | U (83) = 266.5, p      |
| Median (IQR)      | 6 (7.5)             | 2 (4)                    | < .001                 |
| <b>Anxiety</b>    |                     |                          |                        |
| Mean (SD)         | 9.7 (3.9)           | 5.3 (3.2)                | t (81) = -5.58, p      |
|                   |                     |                          | < .001                 |

SD = standard deviation; IQR = interquartile range; DS = dissociative seizures

**Table 10. Multiscale Dissociation Inventory T-scores (MDI) and Somatoform Dissociation Questionnaire scores (SDQ-20)**

|                                      | <b>DS</b>    | <b>Control</b> | <b>Test statistics</b> |
|--------------------------------------|--------------|----------------|------------------------|
| <b>MDI</b>                           | n = 39       | n = 43         |                        |
| <b><i>Disengagement</i></b>          |              |                |                        |
| Mean (SD)                            | 84.2 (18.9)  | 61.2 (11.7)    | U (82) = 258.5,        |
| Median (IQR)                         | 80 (24)      | 60 (16)        | p < .001               |
| <b><i>Depersonalisation</i></b>      |              |                |                        |
| Mean (SD)                            | 87.9 (41.2)  | 51.6 (9.1)     | U (82) = 360, p        |
| Median (IQR)                         | 82 (62)      | 47 (9)         | < .001                 |
| <b><i>Derealisation</i></b>          |              |                |                        |
| Mean (SD)                            | 79.9 (27.7)  | 52.8 (8.5)     | U (82) = 296.5,        |
| Median (IQR)                         | 68 (44)      | 46 (11)        | p < .001               |
| <b><i>Emotional Constriction</i></b> |              |                |                        |
| Mean (SD)                            | 67.02 (20.9) | 51.7 (11.8)    | U (82) = 447.5,        |
| Median (IQR)                         | 63 (38)      | 46 (4)         | p < .001               |
| <b><i>Memory Disturbance</i></b>     |              |                |                        |
| Mean (SD)                            | 93.4 (30.7)  | 55.2 (11.9)    | U (82) = 211, p        |
| Median (IQR)                         | 90 (57)      | 52 (19)        | < .001                 |
| <b><i>Identity Dissociation</i></b>  |              |                |                        |
| Mean (SD)                            | 78 (48.2)    | 50.9 (9.3)     | U (82) = 537, p        |
| Median (IQR)                         | 47 (47)      | 47 (0)         | < .001                 |
| <b>SDQ-20</b>                        | n = 37       | n = 43         |                        |
| Mean (SD)                            | 34.2 (7.2)   | 21.9 (3.6)     | U (80) = 59, p <       |
| Median (IQR)                         | 34 (8)       | 21 (2)         | .001                   |

SD = standard deviation; IQR = interquartile range; DS = dissociative seizures

### ***Inventory of Altered Self-Capacities (IASC)***

Table 11 displays the statistical values relevant to the IASC. Patients with DS received higher scores than control participants on the following subscales: Identity Impairment, Abandonment Concerns, Affect Dysregulation, and Tension Reduction Activities.



**Table 11. Inventory of Altered Self-Capacities (IASC) T-scores**

|                                            | <b>DS</b><br>n = 37 | <b>Control</b><br>n = 43 | <b>Test statistics</b><br>unadjusted<br>(adjusted where<br>different) |
|--------------------------------------------|---------------------|--------------------------|-----------------------------------------------------------------------|
| <b><i>Interpersonal Conflict</i></b>       |                     |                          |                                                                       |
| Mean (SD)                                  | 71 (15.8)           | 63.9 (12.5)              | t (78) = -2.21, p = .03 (p = .09)                                     |
| <b><i>Idealisation-Disillusionment</i></b> |                     |                          |                                                                       |
| Mean (SD)                                  | 65.4 (15.4)         | 61.2 (13.8)              | U (80) = 662.5, p = .199 (p = .398)                                   |
| Median (IQR)                               | 68 (24.5)           | 58 (17)                  |                                                                       |
| <b><i>Abandonment Concerns</i></b>         |                     |                          |                                                                       |
| Mean (SD)                                  | 70.5 (18.7)         | 57.4 (12.9)              | U (80) = 457, p = .001 (p = .004)                                     |
| Median (IQR)                               | 67 (36)             | 51 (16)                  |                                                                       |
| <b><i>Identity Impairment</i></b>          |                     |                          |                                                                       |
| Mean (SD)                                  | 71.9 (17.3)         | 59.8 (14.3)              | U (80) = 466, p = .001 (p = .004)                                     |
| Median (IQR)                               | 72 (31.5)           | 54 (17)                  |                                                                       |
| <b><i>Susceptibility to Influence</i></b>  |                     |                          |                                                                       |
| Mean (SD)                                  | 64.7 (18.2)         | 60.5 (12.8)              | U (80) = 733.5, p = .548                                              |
| Median (IQR)                               | 62 (29)             | 59 (17)                  |                                                                       |
| <b><i>Affect Dysregulation</i></b>         |                     |                          |                                                                       |
| Mean (SD)                                  | 79.5 (18.4)         | 61.3 (13.4)              | U (80) = 348, p < .001                                                |
| Median (IQR)                               | 77 (34.5)           | 56 (22)                  |                                                                       |
| <b><i>Tension Reduction Activities</i></b> |                     |                          |                                                                       |
| Mean (SD)                                  | 73.03 (19.5)        | 59.2 (12.1)              | U (80) = 465, p = .001 (p = .004)                                     |
| Median (IQR)                               | 68 (38)             | 57 (19)                  |                                                                       |

DS = dissociative seizures; SD = standard deviation; IQR = interquartile range

### ***Ictal symptoms***

When asked about their most recent seizures, the most frequently reported symptoms by patients with DS were cognitive and autonomic arousal phenomena. With regards to their most severe seizure, the most frequent symptoms were mental state, autonomic arousal and cognitive symptoms. In fact, during the most severe seizure, 100% of patients reported cognitive symptoms. Table 12 displays the proportion of patients (n, %) reporting at least one of each symptom type, and the average number (mean, SD) of each symptom type across the whole DS group.

**Table 12. Proportion of DS patients reporting at least one symptom for each ictal symptom type, and the average number of each symptom type reported across all patients**

| <b>n = 36</b>             | <b>Most recent</b> | <b>Most severe</b> |
|---------------------------|--------------------|--------------------|
| <b>Ictal symptom type</b> | <b>seizure</b>     | <b>seizure</b>     |
| <b>Chest / abdomen</b>    |                    |                    |
| n (%)                     | 25 (69.4%)         | 29 (80.6%)         |
| Mean (SD)                 | 1.08 (.94)         | 1.86 (1.4)         |
| <b>Mental state</b>       |                    |                    |
| n (%)                     | 29 (80.6%)         | 33 (91.7%)         |
| Mean (SD)                 | 2.53 (1.8)         | 3.36 (1.6)         |
| <b>Autonomic arousal</b>  |                    |                    |
| n (%)                     | 32 (88.9%)         | 33 (91.7%)         |
| Mean (SD)                 | 2.25 (1.3)         | 2.58 (1.4)         |
| <b>General symptoms</b>   |                    |                    |
| n (%)                     | 24 (66.7%)         | 27 (75%)           |
| Mean (SD)                 | .917 (.77)         | 1.19 (.822)        |
| <b>Cognitive</b>          |                    |                    |
| n (%)                     | 34 (94.4%)         | 36 (100%)          |
| Mean (SD)                 | 3.31 (2.87)        | 5.19 (2.68)        |

SD = standard deviation; max = maximum

#### **5.3.4. Exploratory correlational analyses**

Correlations between the subscales of any one measure are not reported because these are predictable and of limited value in the current analyses. Correlations that were significant at the specified alpha level ( $p < .01$ ) in the DS group are reported in the tables below; however, when a significant relationship between two measures was observed in both groups, the finding is not discussed further in the text, due to the lack of explanatory value specific to the clinical group.

#### **TEC**

Significant correlations for the TEC subscales can be found in Table 13. TEC Total scores were positively correlated with MDI Depersonalisation and Emotional Constriction scores. Furthermore, TEC mean impact scores were positively associated with HADS Depression scores. Sexual abuse scores were positively

associated with MDI Identity Dissociation, and TEC physical abuse scores positively correlated with IASC Abandonment Concerns scores. In the traumatised group, there was a significant correlation between PDS Arousal scores and TEC physical abuse scores.

**Table 13. Traumatic Experiences Checklist (TEC) correlations**

| Variables                   | DS |         |          | Control |         |          |
|-----------------------------|----|---------|----------|---------|---------|----------|
|                             | n  | r / rho | p-value  | n       | r / rho | p-value  |
| <b>TEC total</b>            |    |         |          |         |         |          |
| MDI Depersonalisation*      | 39 | .444    | p = .005 | 43      | .226    | p = .145 |
| MDI Emotional Constriction* | 39 | .433    | p = .006 | 43      | .312    | p = .041 |
| <b>TEC mean impact</b>      |    |         |          |         |         |          |
| HADS Depression*            | 39 | .434    | p = .006 | 42      | .090    | p = .571 |
| <b>TEC sexual abuse</b>     |    |         |          |         |         |          |
| MDI Identity Dissociation*  | 39 | .483    | p = .002 | 43      | .090    | p = .567 |
| <b>TEC physical abuse</b>   |    |         |          |         |         |          |
| IASC Abandonment Concerns*  | 37 | .505    | p = .001 | 43      | .078    | p = .620 |
| PDS Arousal*                | 29 | .482    | p = .008 | 28      | .331    | p = .085 |

IASC = Inventory of Altered Self-Capacities; PDS = Post-traumatic diagnostic scale; HADS = Hospital Anxiety & Depression Scale; MDI = Multiscale Dissociation Inventory; DS = dissociative seizures

\* denotes correlation significant in the DS group only (p < .01)

## PDS

Significant correlations between scores on the PDS and other variables are shown in Table 14. In this subgroup of participants (those reporting at least moderate impact of one adverse life event on the TEC), PDS Total scores were positively correlated with HADS Anxiety and Depression scores, IASC Abandonment Concerns and Tension Reduction Activities. Furthermore, PDS Total scores were also positively associated with ictal cognitive symptoms during patients' most recent seizure. PDS Arousal scores were positively associated with HADS Anxiety and Depression scores and IASC Tension Reduction Activities. Moreover, PDS Avoidance scores were associated (positively) with a number of variables, including: HADS Depression scores, IASC Abandonment Concerns and Tension Reduction Activities, and MDI Disengagement and Memory Disturbance. Finally, PDS Re-experiencing symptoms were positively associated with total ictal symptoms during patients' most recent seizures.

**Table 14. Post-traumatic Diagnostic Scale (PDS) correlations**

| Variables                          | DS |         |          | Control |         |          |
|------------------------------------|----|---------|----------|---------|---------|----------|
|                                    | n  | r / rho | p-value  | n       | r / rho | p-value  |
| <b>PDS Total</b>                   |    |         |          |         |         |          |
| HADS Anxiety*                      | 29 | .498    | p = .006 | 28      | .388    | p = .041 |
| HADS Depression*                   | 29 | .592    | p = .001 | 28      | .432    | p = .022 |
| IASC Abandonment Concerns*         | 28 | .574    | p = .001 | 28      | .379    | p = .046 |
| IASC Tension Reduction Activities* | 28 | .538    | p = .003 | 28      | .274    | p = .159 |
| Ictal cognitive symptoms (recent)* | 27 | .524    | p = .005 |         |         |          |
| <b>PDS Arousal</b>                 |    |         |          |         |         |          |
| HADS Anxiety*                      | 29 | .608    | p < .001 | 28      | .415    | p = .028 |
| HADS Depression*                   | 29 | .552    | p = .002 | 28      | .384    | p = .044 |
| IASC Tension Reduction Activities* | 28 | .515    | p = .005 | 28      | .377    | p = .048 |
| <b>PDS Avoidance</b>               |    |         |          |         |         |          |
| HADS Depression*                   | 29 | .628    | p < .001 | 28      | .349    | p = .069 |
| IASC Abandonment Concerns*         | 28 | .579    | p = .001 | 28      | .270    | p = .164 |
| IASC Tension Reduction Activities* | 28 | .677    | p < .001 | 28      | .152    | p = .439 |
| MDI Disengagement*                 | 29 | .478    | p = .009 | 28      | .013    | p = .949 |
| MDI Memory Disturbance*            | 29 | .520    | p = .004 | 28      | .105    | p = .594 |
| <b>PDS Re-experiencing</b>         |    |         |          |         |         |          |
| Ictal total symptoms (recent)*     | 27 | .506    | .007     |         |         |          |

IASC = Inventory of Altered Self-Capacities; HADS = Hospital Anxiety & Depression Scale;

MDI = Multiscale Dissociation Inventory; DS = dissociative seizures

\* denotes correlation significant in the DS group only (p < .01)

## HADS

Statistical values for the correlations involving the two HADS subscales are presented in Table 15. In addition to the associations already mentioned, scores on the Anxiety subscale of the HADS positively correlated with IASC Abandonment Concerns and ictal chest/abdomen symptoms (most recent seizure). Moreover, the HADS Depression subscale was positively correlated with IASC Affect Dysregulation and Tension Reduction Activities, and MDI Memory Disturbance.

**Table 15. Hospital Anxiety & Depression Scale (HADS) correlations**

| Variables                   | DS |            |          | Control |            |          |
|-----------------------------|----|------------|----------|---------|------------|----------|
|                             | n  | r /<br>rho | p-value  | n       | r /<br>rho | p-value  |
| <b>HADS Anxiety</b>         |    |            |          |         |            |          |
| IASC Abandonment Concerns*  | 37 | .458       | p = .004 | 43      | .385       | p = .011 |
| IASC Affect Dysregulation   | 37 | .545       | p < .001 | 43      | .607       | p < .001 |
| Ictal CA symptoms (recent)* | 36 | .464       | p = .004 |         |            |          |
| <b>HADS Depression</b>      |    |            |          |         |            |          |
| IASC Affect Dysregulation*  | 37 | .503       | p = .001 | 43      | .384       | p = .011 |
| IASC Tension Reduction      |    |            |          |         |            |          |
| Activities*                 | 37 | .512       | p = .001 | 43      | .248       | p = .109 |
| MDI Memory Disturbance*     | 39 | .523       | p = .001 | 43      | .022       | p = .886 |

IASC = Inventory of Altered Self-Capacities; MDI = Multiscale Dissociation Inventory;

DS = Dissociative seizures; CA symptoms = chest / abdomen symptoms

\* denotes correlation significant in the DS group only (p < .01)

### **MDI**

Significant correlations for the MDI can be found in Table 16. Several of the MDI subscales were positively associated with IASC Tension Reduction Activities, including Depersonalisation, Derealisation, Emotional Constriction, and Identity Dissociation. In addition, the MDI Depersonalisation subscale was positively associated with ictal mental state symptoms (most recent and most severe seizures) and the total number of ictal symptoms (most recent seizure). Moreover, MDI Derealisation scores were also positively correlated with ictal mental state symptoms (most recent and most severe seizures). Finally, scores on the MDI Identity Dissociation subscale were positively correlated with ictal cognitive symptoms during patients' most severe seizures.

### **SDQ-20**

SDQ-20 scores were not significantly associated with any other variable measured at the desired alpha level, with the seizure-related variable included or excluded.

**Table 16. Multiscale Dissociation Inventory (MDI) correlations**

| Variables                             | DS |       |          | Control |       |          |
|---------------------------------------|----|-------|----------|---------|-------|----------|
|                                       | n  | r/rho | p-value  | n       | r/rho | p-value  |
| <b>MDI Depersonalisation</b>          |    |       |          |         |       |          |
| Ictal mental state symptoms (recent)* | 36 | .649  | p < .001 |         |       |          |
| Ictal mental state symptoms (severe)* | 36 | .616  | p < .001 |         |       |          |
| Ictal total symptoms (recent)*        | 36 | .497  | p = .002 |         |       |          |
| IASC Tension Reduction Activities*    | 37 | .436  | p = .007 | 43      | .328  | p = .032 |
| <b>MDI Derealisation</b>              |    |       |          |         |       |          |
| Ictal mental state symptoms (recent)* | 36 | .606  | p < .001 |         |       |          |
| Ictal mental state symptoms (severe)* | 36 | .501  | p = .002 |         |       |          |
| IASC Tension Reduction Activities*    | 37 | .487  | p = .002 | 43      | .342  | p = .025 |
| <b>MDI Disengagement</b>              |    |       |          |         |       |          |
| IASC Tension Reduction Activities     | 37 | .638  | p < .001 | 43      | .436  | p = .003 |
| <b>MDI Emotional Constriction</b>     |    |       |          |         |       |          |
| IASC Tension Reduction Activities*    | 37 | .556  | p < .001 | 43      | .038  | p = .810 |
| <b>MDI Memory Disturbance</b>         |    |       |          |         |       |          |
| IASC Tension Reduction Activities     | 37 | .427  | p = .008 | 43      | .418  | p = .005 |
| <b>MDI Identity Dissociation</b>      |    |       |          |         |       |          |
| IASC Tension Reduction Activities*    | 37 | .470  | p = .003 | 43      | .248  | p = .109 |
| Ictal cognitive symptoms (severe)*    | 36 | .459  | p = .005 |         |       |          |

DS = dissociative seizures; IASC = Inventory of Altered Self-Capacities

\* denotes correlation significant in the DS group only (p < .01)

## **IASC**

The statistics for the correlations of IASC subscales are displayed in Tables 13-16 above. All of the significant correlations for this measure have been described in previous sections.

### **5.3.5. Exploratory regression analyses**

#### ***Adverse life experiences & group membership***

The first set of logistic regression analyses were carried out to examine the extent to which adverse life events would differentiate between patients with DS and healthy controls. In the initial univariate logistic regression analyses, TEC mean impact scores, sexual abuse and physical abuse significantly differentiated DS patients from controls (see Table 17), after controlling for YoE. Next, a hierarchical multivariate logistic regression was carried out, involving the following procedure: YoE was entered as the initial predictor variable in block 1, with the TEC variables (mean impact, sexual abuse, and physical abuse scores) entered simultaneously in block 2. Once all variables had been entered, the overall model was highly significant (see Table 18) and correctly classified 70% of cases. The only variable to retain significance in the final step was the reported experience of sexual abuse. The presence of reported sexual abuse was associated with a three-fold increase (Odds Ratio = 3.59) in the likelihood of being diagnosed with DS.

#### ***Psychological variables and group membership***

The second set of logistic regression analyses was carried out to establish the extent to which relevant psychological variables (see below) differentiated patients with DS from healthy controls, whilst controlling for YoE.

##### ***Anxiety and depression***

Both HADS Anxiety and HADS Depression scores were found to be significant predictors of DS group membership, when assessed with univariate logistic regressions (controlling for YoE – see Table 19). However, when entered simultaneously in block 2 of a hierarchical multivariate logistic regression model (YoE as block 1), only YoE and HADS depression scores remained significant (see Table

20). This model successfully classified 73.5% of cases, and each unit increase in HADS Depression scores increased the likelihood of a DS diagnosis by 1.45 times.

**Table 17. Individual logistic regression statistics for the independent prediction of TEC variables on DS group membership, with YoE as covariate**

| n = 82                                       | X <sup>2</sup> | Wald | df | p-value  | Odds Ratio (OR) | Confidence Interval (CI, 95%) |
|----------------------------------------------|----------------|------|----|----------|-----------------|-------------------------------|
| <b>TEC total scores</b>                      |                |      |    |          |                 |                               |
| Block 1:                                     | 3.78           |      | 1  | p = .052 |                 |                               |
| YoE                                          |                | 3.58 | 1  | p = .058 | .84             | .706-1.01                     |
| Block 2:                                     | 0.53           |      | 2  | p = .021 |                 |                               |
| YoE                                          |                | 4.25 | 1  | p = .039 | .82             | .682-.990                     |
| TEC total                                    |                | 4.69 | 1  | p = .03  | 1.12            | 1.01-1.25                     |
| Model                                        | 9.1            |      | 2  | p = .011 |                 |                               |
| <b>TEC mean impact scores</b>                |                |      |    |          |                 |                               |
| Block 1:                                     | 3.41           |      | 1  | p = .065 |                 |                               |
| YoE                                          |                | 3.25 | 1  | p = .072 | .85             | .712-1.01                     |
| Block 2:                                     | 10.1           |      | 1  | p = .001 |                 |                               |
| YoE                                          |                | 2.04 |    | p = .153 | .87             | .722-1.05                     |
| TEC mean impact*                             |                | 8.21 | 1  | p = .004 | 2.56            | 1.35-4.88                     |
| Model                                        | 13.5           |      | 2  | p = .001 |                 |                               |
| <b>TEC sexual abuse (presence/absence)</b>   |                |      |    |          |                 |                               |
| Block 1:                                     | 3.39           |      | 1  | p = .066 |                 |                               |
| YoE                                          |                | 3.23 | 1  | p = .072 | .85             | .712-1.02                     |
| Block 2:                                     | 11.8           |      | 1  | p = .001 |                 |                               |
| YoE                                          |                | 2.78 | 1  | p = .096 | .85             | .702-1.03                     |
| TEC sexual abuse*                            |                | 10.6 | 1  | p = .001 | 5.72            | 2-16.4                        |
| Model                                        | 15.2           |      | 2  | p < .001 |                 |                               |
| <b>TEC physical abuse (presence/absence)</b> |                |      |    |          |                 |                               |
| Block 1:                                     | 3.78           |      | 1  | p = .052 |                 |                               |
| YoE                                          |                | 3.58 | 1  | p = .058 | .84             | .706-1.01                     |
| Block 2:                                     | 7.84           |      | 1  | p = .005 |                 |                               |
| YoE                                          |                | 4.28 | 1  | p = .039 | .82             | .676-.989                     |
| TEC physical abuse*                          |                | 7.34 | 1  | p = .007 | 3.73            | 1.44-9.65                     |
| Model                                        | 11.6           |      | 2  | p = .003 |                 |                               |

TEC = Traumatic Experiences Checklist; YoE = years of full-time education (or equiv.)

\* denotes significant variables (p < .01)



**Table 18. Hierarchical logistic regression statistics for the predictive value of TEC variables on DS group membership, with YoE as covariate**

| <b>n = 80</b>      | <b>X<sup>2</sup></b> | <b>Wald</b> | <b>df</b> | <b>p-value</b> | <b>Odds Ratio (OR)</b> | <b>Confidence Interval (CI, 95%)</b> |
|--------------------|----------------------|-------------|-----------|----------------|------------------------|--------------------------------------|
| <b>Block 1:</b>    | 3.05                 |             | 1         | p = .081       |                        |                                      |
| YoE                |                      | 2.91        | 1         | p = .088       | .857                   | .718-1.02                            |
| <b>Block 2:</b>    | 18.3                 |             | 3         | p < .001       |                        |                                      |
| YoE                |                      | 2.51        | 1         | p = .113       | .845                   | .687-1.04                            |
| TEC mean impact    |                      | 3.71        | 1         | p = .054       | 1.05                   | 1.01-1.09                            |
| TEC sexual abuse*  |                      | 5.04        | 1         | p = .025       | 3.59                   | 1.18-10.96                           |
| TEC physical abuse |                      | 1.56        | 1         | p = .211       | 1.97                   | .681-5.69                            |
| Model              | 21.3                 |             | 4         | p < .001       |                        |                                      |

TEC = Traumatic Experiences Checklist; YoE = years of full-time education (or equiv.)

\* denotes significant variables (p < .05)

**Table 19. Individual logistic regression statistics for the differentiation of group membership by HADS anxiety and depression scores, with YoE as covariate**

| <b>n = 83</b>                 | <b>X<sup>2</sup></b> | <b>Wald</b> | <b>df</b> | <b>p-value</b> | <b>Odds Ratio (OR)</b> | <b>Confidence Interval (CI, 95%)</b> |
|-------------------------------|----------------------|-------------|-----------|----------------|------------------------|--------------------------------------|
| <b>HADS Anxiety scores</b>    |                      |             |           |                |                        |                                      |
| Block 1                       | 4.17                 |             | 1         | p = .041       |                        |                                      |
| YoE                           |                      | 3.94        | 1         | p = .047       | .84                    | .701-.998                            |
| Block 2                       | 27.7                 |             | 1         | p < .001       |                        |                                      |
| YoE                           |                      | 3.79        | 1         | p = .051       | .8                     | .643-1                               |
| HADS Anxiety*                 |                      | 16.5        | 1         | p < .001       | 1.44                   | 1.21-1.72                            |
| Model                         | 31.9                 |             | 2         | p < .001       |                        |                                      |
| <b>HADS Depression scores</b> |                      |             |           |                |                        |                                      |
| Block 1                       | 4.17                 |             | 1         | p = .041       |                        |                                      |
| YoE                           |                      | 3.94        | 1         | p = .047       | .84                    | .701-.998                            |
| Block 2                       | 35.7                 |             | 1         | p < .001       |                        |                                      |
| YoE                           |                      | 4.86        | 1         | p = .027       | .76                    | .599-.970                            |
| HADS Depression*              |                      | 18.1        | 1         | p < .001       | 1.64                   | 1.3-2.06                             |
| Model                         | 39.9                 |             | 2         | p < .001       |                        |                                      |

HADS = Hospital Anxiety & Depression Scale; YoE = years of full-time education (or equiv.)

\* denotes significant variables (p < .01)

**Table 20. Hierarchical logistic regression statistics for the prediction of DS group membership by HADS Depression and Anxiety scores, with YoE as covariate**

| <b>n = 83</b>    | <b>X<sup>2</sup></b> | <b>Wald</b> | <b>df</b> | <b>p-value</b> | <b>Odds Ratio (OR)</b> | <b>Confidence Interval (CI, 95%)</b> |
|------------------|----------------------|-------------|-----------|----------------|------------------------|--------------------------------------|
| <b>Block 1</b>   | 4.17                 |             | 1         | p = .041       |                        |                                      |
| YoE              |                      | 3.94        | 1         | p = .047       | .84                    | .701-.998                            |
| <b>Block 2</b>   | 37.6                 |             | 2         | p < .001       |                        |                                      |
| YoE*             |                      | 4.37        | 1         | p = .037       | .77                    | .599-.984                            |
| HADS Anxiety     |                      | 1.73        | 1         | p = .188       | 1.16                   | .931-1.44                            |
| HADS Depression* |                      | 7.27        | 1         | p = .007       | 1.45                   | 1.11-1.9                             |
| <b>Model</b>     | 41.8                 |             | 3         | p < .001       |                        |                                      |

HADS = Hospital Anxiety & Depression Scale; YoE = years of full-time education (or equiv.)

\* denoted significant variables (p < .05)

#### *Dissociation*

SDQ-20 scores and scores on all of the MDI subscales were significant predictors of DS group membership, when assessed with univariate logistic regressions (with YoE as covariate, see Tables 21 and 22). SDQ-20 scores remained significant when the analysis was rerun with the seizure-related item removed.

**Table 21. Logistic regression statistics for SDQ-20 scores predicting DS group membership, with YoE as covariate**

| <b>n = 80</b>  | <b>X<sup>2</sup></b> | <b>Wald</b> | <b>df</b> | <b>p-value</b> | <b>Odds Ratio (OR)</b> | <b>Confidence Interval (CI, 95%)</b> |
|----------------|----------------------|-------------|-----------|----------------|------------------------|--------------------------------------|
| <b>Block 1</b> | 3.18                 |             | 1         | p = .075       |                        |                                      |
| YoE            |                      | 3.03        | 1         | p = .082       | .854                   | .714-1.02                            |
| <b>Block 2</b> | 63.96                |             | 1         | p < .001       |                        |                                      |
| YoE            |                      | .389        | 1         | p = .533       | .911                   | .678-1.22                            |
| SDQ-20*        |                      | 22.7        | 1         | p < .001       | 1.571                  | 1.3-1.89                             |
| <b>Model</b>   | 67.1                 |             | 1         | p < .001       |                        |                                      |

SDQ-20: Somatoform Dissociation Questionnaire – 20 item version; YoE = years of full-time education (or equiv.)

\* denotes significant variables (p < .05)

**Table 22. Individual logistic regression statistics for the predictive power of each MDI subscale scores on DS diagnosis, with YoE as covariate**

| <b>n = 82</b>          | <b>X<sup>2</sup></b> | <b>Wald</b> | <b>df</b> | <b>p-value</b> | <b>Odds Ratio (OR)</b> | <b>Confidence Interval (CI, 95%)</b> |
|------------------------|----------------------|-------------|-----------|----------------|------------------------|--------------------------------------|
| <b>MDI DENG</b>        |                      |             |           |                |                        |                                      |
| Block 1                | 3.78                 |             | 1         | p = .052       |                        |                                      |
| YoE                    |                      | 3.58        | 1         | p = .058       | .84                    | .706-1.01                            |
| Block 2                | 36.5                 |             | 1         | p < .001       |                        |                                      |
| YoE                    |                      | 4.06        | 1         | p = .044       | .79                    | .625-.994                            |
| MDI DENG*              |                      | 20.5        | 1         | p < .001       | 1.1                    | 1.06-1.15                            |
| Model                  | 40.3                 |             | 2         | p < .001       |                        |                                      |
| <b>MDI DEPR</b>        |                      |             |           |                |                        |                                      |
| Block 1 (see MDI DENG) |                      |             |           |                |                        |                                      |
| Block 2                | 32.0                 |             | 1         | p < .001       |                        |                                      |
| YoE                    |                      | 2.35        | 1         | p = .126       | .84                    | .674-1.05                            |
| MDI DEPR*              |                      | 13          | 1         | p < .001       | 1.07                   | 1.03-1.12                            |
| Model                  | 35.8                 |             | 2         | p < .001       |                        |                                      |
| <b>MDI DERL</b>        |                      |             |           |                |                        |                                      |
| Block 1 (see MDI DENG) |                      |             |           |                |                        |                                      |
| Block 2                | 33                   |             | 1         | p < .001       |                        |                                      |
| YoE                    |                      | 1.55        | 1         | p = .213       | .87                    | .692-1.09                            |
| MDI DERL*              |                      | 14.6        | 1         | p < .001       | 1.1                    | 1.05-1.15                            |
| Model                  | 36.8                 |             | 2         | p < .001       |                        |                                      |
| <b>MDI ECON</b>        |                      |             |           |                |                        |                                      |
| Block 1 (see MDI DENG) |                      |             |           |                |                        |                                      |
| Block 2                | 17.4                 |             | 1         | p < .001       |                        |                                      |
| YoE                    |                      | 4.71        | 1         | p = .03        | .8                     | .655-.979                            |
| MDI ECON*              |                      | 11.6        | 1         | p = .001       | 1.06                   | 1.03-1.11                            |
| Model                  | 21.2                 |             | 2         | p < .001       |                        |                                      |
| <b>MDI MEMD</b>        |                      |             |           |                |                        |                                      |
| Block 1 (see MDI DENG) |                      |             |           |                |                        |                                      |
| Block 2                | 42.2                 |             | 1         | p < .001       |                        |                                      |
| YoE                    |                      | 1.37        | 1         | p = .242       | .86                    | .677-1.1                             |
| MDI MEMD*              |                      | 17.5        | 1         | p < .001       | 1.08                   | 1.04-1.13                            |
| Model                  | 46                   |             | 2         | p < .001       |                        |                                      |
| <b>MDI IDDIS</b>       |                      |             |           |                |                        |                                      |
| Block 1 (see MDI DENG) |                      |             |           |                |                        |                                      |
| Block 2                | 16                   |             | 1         | p < .001       |                        |                                      |
| YoE                    |                      | 2.95        | 1         | p = .086       | .84                    | .695-1.02                            |
| MDI IDDIS*             |                      | 7.4         | 1         | p = .007       | 1.05                   | 1.01-1.09                            |
| Model                  | 19.8                 |             | 1         | p < .001       |                        |                                      |

MDI = Multiscale Dissociation Inventory; DENG = Disengagement; DEPR = Depersonalisation; ECON = Emotion Constriction; MEMD = Memory Disturbance; IDDIS = Identity Dissociation; YoE = years of full-time education (or equiv.)

\* denotes significant variables (p < .01)

It was of interest to assess the relative predictive power of these different dissociative symptoms on group status. However, due to power considerations (see Chapter 4, section 4.5.2), only two of the MDI subscales were selected for entry into the multivariate analysis along with SDQ-20 scores and YoE. MDI Depersonalisation and Derealisation scores had previously been found to be correlated with ictal DS symptoms (mental state); therefore, these subscales were selected due to their potential significance to the occurrence of seizures in the DS group.

Scores from the MDI Depersonalisation and Derealisation subscales were entered simultaneously with SDQ-20 scores in block 2 of a hierarchical logistic regression (block 1 = YoE), with group status as the outcome variable. The final model was highly significant, indicating that dissociation was a strong predictor of DS diagnosis (see Table 23), successfully categorising 92.5% of cases. However, only SDQ-20 scores remained significant with all variables included in the model. When the analysis was run again, excluding the seizure-related item from the SDQ scores, the same results occurred as in the initial analysis, although the model correctly classified 86.3% of cases in this second analysis.

**Table 23. Hierarchical logistic regression statistics for the predictive value of MDI Depersonalisation, Derealisation and SDQ-20 scores on group status, with YoE as covariate**

| <b>n = 80</b>  | <b>X<sup>2</sup></b> | <b>Wald</b> | <b>df</b> | <b>p-value</b>     | <b>Odds Ratio (OR)</b> | <b>Confidence Interval (CI, 95%)</b> |
|----------------|----------------------|-------------|-----------|--------------------|------------------------|--------------------------------------|
| <b>Block 1</b> | 3.18                 |             | 1         | p = .075           |                        |                                      |
| YoE            |                      | 3.03        | 1         | p = .082           | .854                   | .714-1.02                            |
| <b>Block 2</b> | 73.5                 |             | 3         | p < .001           |                        |                                      |
| YoE            |                      | .121        | 1         | p = .728           | 1.06                   | .762-1.48                            |
| MDI DEPR       |                      | .03         | 1         | p = .862           | .993                   | .921-1.07                            |
| MDI DERL       |                      | 2.88        | 1         | p = .09            | 1.1                    | .985-1.23                            |
| SDQ-20*        |                      | 15.8        | 1         | p < .001           | 1.53                   | 1.24-1.89                            |
| <b>Model</b>   | <b>76.7</b>          |             | <b>4</b>  | <b>p &lt; .001</b> |                        |                                      |

MDI = Multiscale Dissociation Inventory; DEPR = Depersonalisation; DERL = Derealisation;

SDQ-20 = Somatoform Dissociation Questionnaire – 20 item version; YoE = years of full-time education (or equiv.)

\* denotes significant variables (p < .05)

### Borderline personality traits

Univariate logistic regression analyses (with YoE as covariate) showed that all of the IASC subscales were significant independent predictors of DS diagnosis (see Table 24).

**Table 24. Individual logistic regression statistics for the predictive power of IASC subscale scores on DS diagnosis, with YoE as covariate**

| <b>n = 80</b>                | <b>X<sup>2</sup></b> | <b>Wald</b> | <b>df</b> | <b>p-value</b> | <b>Odds Ratio (OR)</b> | <b>Confidence Interval (CI, 95%)</b> |
|------------------------------|----------------------|-------------|-----------|----------------|------------------------|--------------------------------------|
| <b>IASC AC</b>               |                      |             |           |                |                        |                                      |
| <i>Block 1</i>               | 3.18                 |             | 1         | p = .075       |                        |                                      |
| YoE                          |                      | 3.03        | 1         | p = .082       | .85                    | .714-1.02                            |
| <i>Block 2</i>               | 14                   |             | 1         | p < .001       |                        |                                      |
| YoE                          |                      | 4.3         | 1         | p = .038       | .81                    | .661-.988                            |
| IASC AC*                     |                      | 11.1        | 1         | p = .001       | 1.06                   | 1.02-1.1                             |
| <i>Model</i>                 | 17.2                 |             | 2         | p < .001       |                        |                                      |
| <b>IASC II</b>               |                      |             |           |                |                        |                                      |
| <i>Block 1 (see IASC AC)</i> |                      |             | 2         | p < .001       |                        |                                      |
| YoE                          |                      |             |           |                |                        |                                      |
| <i>Block 2</i>               | 11.5                 |             | 1         | p = .001       |                        |                                      |
| YoE                          |                      | 3.5         | 1         | p = .061       | .83                    | .685-1.01                            |
| IASC II*                     |                      | 9.83        | 1         | p = .002       | 1.05                   | 1.02-1.09                            |
| <i>Model</i>                 | 14.7                 |             | 2         | p = .001       |                        |                                      |
| <b>IASC AD</b>               |                      |             |           |                |                        |                                      |
| <i>Block 1 (see IASC AC)</i> |                      |             |           |                |                        |                                      |
| YoE                          |                      |             |           |                |                        |                                      |
| <i>Block 2</i>               | 22.8                 |             | 1         | p < .001       |                        |                                      |
| YoE                          |                      | 3.92        | 1         | p = .048       | .81                    | .662-.998                            |
| IASC AD*                     |                      | 16.5        | 1         | p < .001       | 1.07                   | 1.04-1.11                            |
| <i>Model</i>                 | 26                   |             | 2         | p < .001       |                        |                                      |
| <b>IASC TRA</b>              |                      |             |           |                |                        |                                      |
| <i>Block 1 (see IASC AC)</i> |                      |             |           |                |                        |                                      |
| YoE                          |                      |             |           |                |                        |                                      |
| <i>Block 2</i>               | 16.9                 |             | 1         | p < .001       |                        |                                      |
| YoE                          |                      | 5.9         | 1         | p = .015       | .77                    | .630-.952                            |
| IASC TRA*                    |                      | 12.6        | 1         | p < .001       | 1.07                   | 1.03-1.1                             |
| <i>Model</i>                 | 20.1                 |             | 2         | p < .001       |                        |                                      |

YoE = years of full-time education (or equiv.); IASC = Inventory of Altered Self-Capacities; AD = Affective Dysregulation; II = Identity Impairment; TRA = Tension Reduction Activities; AC = Abandonment Concerns; DS = dissociative seizures  
 \* denotes significant variables (p < .01)

Due to power considerations, only the IASC subscales that had been found to be correlated with other important variables (e.g. trauma, post-traumatic symptoms, anxiety, depression) were selected for entry into the multivariate analysis (Abandonment Concerns, Affect Dysregulation, Tension Reduction Activities). Scores for the selected IASC subscales were simultaneously entered into block 2, with YoE entered in block 1 and with group status as the criterion variable. The final model was highly significant; correctly identifying 71.3% of cases. Nevertheless, only the coefficients for the IASC AD subscale and YoE remained significant in the final model (see Table 25, Analysis 1).

As both HADS anxiety and depression scores had been noted to be highly correlated with the IASC AD scores in the DS group, it was necessary to investigate the extent to which the predictive relationship of IASC AD to DS diagnosis could be explained by HADS scores. Therefore, a further multivariate logistic regression was run with YoE in block 1, HADS Anxiety and Depression scores entered in block 2, and IASC AD scores entered in block 3. Whilst the final model including these subscales was highly significant, the addition of IASC AD scores in the last block did not significantly improve the fit of the model. Only the HADS depression scores remained significant in the final model, suggesting that the effect of IASC AD on DS diagnosis might be attributable to the raised depression scores in that group (see Table 25, Analysis 2). A mediation analysis was carried out to examine this further. This analysis showed that there was a significant indirect effect of IASC AD scores on group membership through HADS Depression scores ( $b = .045$ ,  $z = 2.74$ ,  $p = .006$ ). With HADS Depression scores included in this model, the direct effect of IASC AD scores to group membership was not significant ( $b = .034$ ,  $z = 1.77$ ,  $p = .077$ ).

### ***Sexual abuse history, depression, somatoform dissociation and group membership***

The first set of analyses reported above indicated that, of the variables relating to adverse life events, a history of sexual abuse was the most important predictor of DS group membership. The second set of analyses showed that, of the possible psychological variables associated with DS, the presence of depression and somatoform dissociation were the unique predictors of the diagnosis.

**Table 25. Hierarchical logistic regression statistics for the predictive relationship of IASC subscales to DS diagnosis, with YoE and HADS scores as covariates**

| <b>n = 80</b>                     | <b>X<sup>2</sup></b> | <b>Wald</b> | <b>df</b> | <b>p-value</b> | <b>Odds Ratio (OR)</b> | <b>Confidence Interval (CI, 95%)</b> |
|-----------------------------------|----------------------|-------------|-----------|----------------|------------------------|--------------------------------------|
| <b>Analysis 1</b>                 |                      |             |           |                |                        |                                      |
| <i>Block 1</i>                    | 3.18                 |             | 1         | p = .075       |                        |                                      |
| YoE                               |                      | 3.03        | 1         | p = .082       | .854                   | .714-1.02                            |
| <i>Block 2</i>                    | 24.6                 |             | 3         | p < .001       |                        |                                      |
| YoE*                              |                      | 4.78        | 1         | p = .029       | .786                   | .634-.975                            |
| IASC AC                           |                      | .115        | 1         | p = .735       | 1.01                   | .961-1.06                            |
| IASC AD*                          |                      | 4.84        | 1         | p = .028       | 1.05                   | 1.01-1.1                             |
| IASC TRA                          |                      | 1.13        | 1         | p = .287       | 1.03                   | .979-1.08                            |
| <i>Model</i>                      | 27.7                 |             | 4         | p < .001       |                        |                                      |
| <b>Analysis 2</b>                 |                      |             |           |                |                        |                                      |
| <i>Block 1 (see Step 1 above)</i> |                      |             |           |                |                        |                                      |
| YoE                               |                      |             |           |                |                        |                                      |
| <i>Block 2</i>                    | 35.2                 |             | 2         | p < .001       |                        |                                      |
| YoE                               |                      | 3.71        | 1         | p = .054       | .783                   | .610-1                               |
| HADS Anxiety                      |                      | 1.73        | 1         | p = .189       | 1.16                   | .932-1.43                            |
| HADS Depression*                  |                      | 6.83        | 1         | p = .009       | 1.43                   | 1.09-1.87                            |
| <i>Model</i>                      | 38.4                 |             | 3         | p < .001       |                        |                                      |
| <i>Block 3</i>                    | 1.46                 |             | 1         | p = .227       |                        |                                      |
| YoE                               |                      | 3.75        | 1         | p = .053       | .783                   | .612-1                               |
| HADS Anxiety                      |                      | .334        | 1         | p = .563       | 1.07                   | .843-1.37                            |
| HADS Depression*                  |                      | 5.53        | 1         | p = .019       | 1.38                   | 1.06-1.81                            |
| IASC AD                           |                      | 1.43        | 1         | p = .232       | 1.03                   | .982-1.08                            |
| <i>Model</i>                      | 39.9                 |             | 4         | p < .001       |                        |                                      |

IASC = Inventory of Altered Self-Capacities; AD = Affective Dysregulation; TRA = Tension Reduction Activities; AC = Abandonment Concerns; HADS = Hospital Anxiety & Depression Scale; YoE = Years of full-time education (or equiv.)  
DS = dissociative seizures  
\* denotes significant variables (p < .05)

A final set of exploratory analyses were, therefore, conducted to examine whether the predictive relationship of self-reported sexual abuse to DS diagnosis might be explained by depression and/or somatoform dissociation scores. Firstly, a multivariate hierarchical logistic regression was conducted, in which the following procedure was implemented: block 1 = YoE, block 2 = SDQ-20, HADS Depression, block 3 = TEC sexual abuse. The statistics for these analyses can be found in Table 26. Within the final model, the only variable that remained significant was the SDQ-20. The overall model was highly significant and allowed accurate classification of 91.1% of cases. This analysis was run again with SDQ-20 scores calculated excluding the seizure-item. A very similar pattern of findings was revealed, with the key difference that in the final model, HADS Depression scores remained significant, along with SDQ-20 scores. Table 27 displays the statistical values for this analysis. This adjusted analysis correctly classified 88.6% of cases.

**Table 26. Hierarchical logistic regression statistics: sexual abuse, somatoform dissociation (SDQ-20), depression (HADS Depression) and DS group membership**

| <b>n = 79</b>    | <b>X<sup>2</sup></b> | <b>Wald</b> | <b>df</b> | <b>p-value</b> | <b>Odds Ratio (OR)</b> | <b>Confidence Interval (CI, 95%)</b> |
|------------------|----------------------|-------------|-----------|----------------|------------------------|--------------------------------------|
| <b>Block 1</b>   | 2.8                  |             | 1         | p = .094       |                        |                                      |
| YoE              |                      | 2.68        | 1         | p = .101       | .861                   | .72-1.03                             |
| <b>Block 2</b>   | 67.03                |             | 2         | p < .001       |                        |                                      |
| YoE              |                      | .286        | 1         | p = .593       | .918                   | .670-1.26                            |
| SDQ-20*          |                      | 15.3        | 1         | p < .001       | 1.46                   | 1.21-1.76                            |
| HADS Depression  |                      | 3.22        | 1         | p = .073       | 1.27                   | .978-1.66                            |
| <b>Model</b>     | 69.8                 |             | 3         | p < .001       |                        |                                      |
| <b>Block 3</b>   | .0                   |             | 1         | p = .997       |                        |                                      |
| YoE              |                      | .285        | 1         | p = .594       | .918                   | .669-1.26                            |
| SDQ-20*          |                      | 15.3        | 1         | p < .001       | 1.46                   | 1.21-1.76                            |
| HADS Depression  |                      | 2.61        | 1         | p = .106       | 1.27                   | .95-1.71                             |
| TEC sexual abuse |                      | .0          | 1         | p = .997       | 1                      | .136-7.42                            |
| <b>Model</b>     | 69.8                 |             | 4         | p < .001       |                        |                                      |

YoE = years of full-time education (or equiv.); SDQ-20 = Somatoform Dissociation Questionnaire – 20 item version; HADS = Hospital Anxiety & Depression Scale; TEC = Traumatic Experiences Checklist

\* denotes significant variables (p < .05)



**Table 27. Hierarchical logistic regression statistics: sexual abuse, somatoform dissociation (SDQ, minus seizure-item), depression (HADS depression) and DS group membership**

| <b>n = 79</b>    | <b>X<sup>2</sup></b> | <b>Wald</b> | <b>df</b> | <b>p-value</b> | <b>Odds Ratio (OR)</b> | <b>Confidence Interval (CI, 95%)</b> |
|------------------|----------------------|-------------|-----------|----------------|------------------------|--------------------------------------|
| <b>Block 1</b>   | 2.8                  |             | 1         | p = .094       |                        |                                      |
| YoE              |                      | 2.68        | 1         | p = .101       | .861                   | .72-1.03                             |
| <b>Block 2</b>   | 53.4                 |             | 2         | p < .001       |                        |                                      |
| YoE              |                      | 1.01        | 1         | p = .314       | .869                   | .660-1.14                            |
| SDQ*             |                      | 11.4        | 1         | p = .001       | 1.33                   | 1.13-1.57                            |
| HADS Depression* |                      | 6.93        | 1         | p = .008       | 1.39                   | 1.09-1.78                            |
| <b>Model</b>     | 56.2                 |             | 3         | p < .001       |                        |                                      |
| <b>Block 3</b>   | .056                 |             | 1         | p = .813       |                        |                                      |
| YoE              |                      | .987        | 1         | p = .321       | .870                   | .661-1.15                            |
| SDQ*             |                      | 11.03       | 1         | p = .001       | 1.33                   | 1.12-1.57                            |
| HADS Depression* |                      | 5.36        | 1         | p = .021       | 1.37                   | 1.05-1.79                            |
| TEC sexual abuse |                      | .057        | 1         | p = .812       | 1.23                   | .230-6.51                            |
| <b>Model</b>     | 56.3                 |             | 4         | p < .001       |                        |                                      |

YoE = years of full-time education (or equiv.); SDQ-20 = Somatoform Dissociation Questionnaire – 20 item version;

HADS = Hospital Anxiety & Depression Scale; TEC = Traumatic Experiences Checklist

\* denotes significant variables (p < .05)

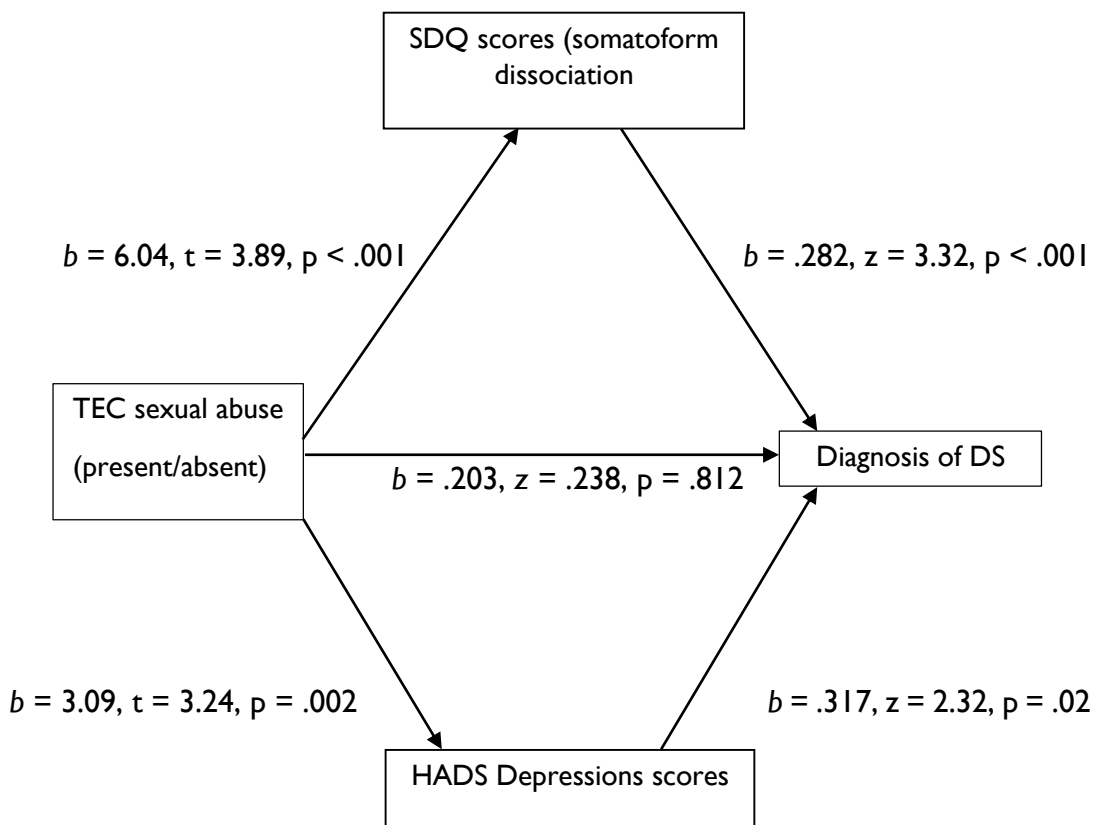
### **Mediation analyses**

The results of the above regression analyses suggested that the relationship between sexual abuse and DS diagnosis might be mediated by somatoform dissociation (SDQ-20 scores) and depressive symptoms (HADS Depression). Therefore, mediation analyses were conducted with TEC sexual abuse (present/absent) as the predictor variable, group (DS, control) as the outcome variable, and SDQ-20 and HADS Depression scores as the mediating variables (with YoE as covariate). In this analysis, TEC sexual abuse significantly predicted SDQ-20 scores ( $b = 7.41$ ,  $t = 4.11$ ,  $p < .001$ ) and HADS Depression scores ( $b = 3.09$ ,  $t = 3.24$ ,  $p = .002$ ). With the mediating variables in the model, the direct effect of TEC sexual abuse on group was not significant ( $b = .004$ ,  $z = .004$ ,  $p = .997$ ). Furthermore, the mediating effect of HADS Depression was not significant ( $b = .746$ ,  $z = 1.39$ ,  $p = .163$ ). On the other hand, the mediating effect of SDQ-20 was significant ( $b = 2.79$ ,  $z = 2.79$ ,  $p = .005$ ).

Once again, the analysis was rerun with the seizure-related item removed from the SDQ scores. In the adjusted analysis, the mediating effect of the SDQ scores

remained significant ( $b = 1.71, z = 2.48, p = .01$ ) but HADS Depression was not a significant mediator ( $b = .977, z = 1.83, p = .068$ ). With HADS Depression and SDQ scores entered in this adjusted analysis, the direct effect of TEC sexual abuse on group was not significant. This final mediation analysis is illustrated in Figure 8.

**Figure 8. Mediation analysis: TEC sexual abuse, SDQ, HADS Depression and DS diagnosis**



## **5.4. Discussion**

### **5.4.1. Summary and interpretation**

#### ***Demographic and clinical characteristics***

The two groups were well-matched on most important demographic variables (e.g. age, gender, ethnic background). However, patients diagnosed with DS reported significantly fewer years of education (YoE) than the comparison group; therefore, this was included as a covariate in subsequent analyses. The patient group reported

taking significantly more prescribed medications than the control group. This finding might be expected due to previous diagnoses of epilepsy and the prevalence of comorbid organic or medically unexplained symptoms/diagnoses in this group. The most common medications reported in the DS group were AEDs, anti-depressants, proton pump inhibitors (PPIs) and analgesics/anti-inflammatories. The two former classes of medication are likely to be related to the occurrence of seizures and depression/anxiety respectively. The frequent prescription of PPIs in this DS sample seems to be related to relatively high rates of gastrointestinal complaints in that group (15%).

Medical diagnoses were also significantly more common in the DS group relative to the control group. In the DS sample, gastrointestinal complaints, hypertension and (a history of) hysterectomy were common. On the other hand, the most common medical diagnosis reported in the control group was asthma. This finding is in accordance with the suggestion that medical trauma or somatic distress may act as a contributing factor to the development of DS. Many of the medical conditions reported by patients with DS were of a chronic/severe nature, and many necessitated surgical or medical interventions. Moreover, common medical comorbidities included conditions that can be exacerbated or precipitated by stress (e.g. hypertension, gastrointestinal diagnoses).

Another marked pattern was that, in female patients, diagnoses associated with reproductive health (e.g. polycystic ovary syndrome, amenorrhea, ovarian cancer) were common. This raises the possibility that for female patients of child-bearing age, threat or harm to the healthy functioning of the reproductive system may act as a significant traumatic/stressful life event, and so might contribute to the development of DS or other dissociative/conversion symptoms.

#### **5.4.2. Adverse life events**

This study has further supported the proposition that DS are associated with high rates of adverse life events. The patient sample reported overall rates of adverse life events that were higher than controls, with significantly greater perceived impact also reported. A wide range of adverse events was endorsed by patients on the TEC.

These findings further confirm the possible importance of general trauma as an aetiological factor in the development and/or precipitation of DS. The findings also indicate that subjective responses to adverse life events (impact ratings) are an important factor in this group.

Interestingly, the total number of adverse life events reported by DS patients (TEC Total scores) correlated positively with scores on two MDI subscales, Emotional Constriction and Depersonalisation. One speculative explanation for this might be that high levels of adversity could contribute to a tendency towards reduced/inhibited emotional experience and detachment from the subjective aspects of experience more generally, in this group. As discussed in Chapter 3, dissociative symptoms such as these might serve a protective psychological function in some respects, by reducing the experienced emotional and subjective impact of adverse life events.

The observed relationship between TEC mean impact and HADS Depression scores suggests that the perceived impact of adverse life events is linked to the more general emotional distress observed in patients with DS. Perhaps higher levels of subjective traumatisation may lead to elevated emotional distress more generally. On the other hand, when rating the perceived subjective impact of life events, patients may have been referring to the impact of life events on their emotional functioning (i.e. depression). An alternative explanation is that the mood dysfunction could be primary, and cause patients to perceive greater impact of life events due to the negative bias associated with such symptoms. In other words, existing mood dysfunction might influence patients' subjective reactions to adversity. One way of examining this relationship further might be to assess patients' views on the impact of life events at different time points, such as before and after commencing treatment with anti-depressant medications. This could provide an opportunity to explore whether the perceived impact of life events improves with amelioration of depressive symptoms.

Regarding types of life event, on the basis of previous research findings, rates of abuse and were specifically examined in the current study. In accordance with previous research and the hypotheses outlined in section 5.1, higher rates of lifetime sexual

and physical abuse were observed in the DS sample, relative to controls. Such experiences, therefore, seem to be important aetiological factors in this disorder, for a significant proportion of patients. When explored further in the regression analyses, self-reported history of sexual abuse was found to be uniquely predictive of DS diagnosis, independently of the perceived impact of all adverse life events (TEC mean impact scores), and a history of physical abuse.

The importance of sexual abuse in the aetiology of DS has been implicated widely in the literature (see Chapter 2, section 2.2.1), and so this study is in line with previous findings. A history of sexual abuse may be a useful indicator of the possibility of DS in patients being assessed with medically intractable seizures. The fact that sexual abuse scores were positively correlated with MDI Identity Dissociation in the current study suggests that higher rates of sexual abuse may be associated with the development of the most severe and disruptive type of dissociative symptom (i.e. alterations in the experience of personal identity). Again, this is in accordance with previous literature which indicates a relationship between dissociative identity disorder and sexual abuse (e.g. Anderson et al., 1993; Ross et al., 1991).

Contrary to expectations, emotional abuse and neglect were not found at higher levels in this sample of patients, relative to healthy controls. As discussed in Chapter 2 (section 2.2.1), previous findings regarding emotional abuse and neglect have been variable, and it has been suggested that such experiences might be mediated by family dysfunction in patients with DS (see Salmon et al., 2003). The lack of group differences in emotional abuse/neglect in the current study could be linked to the fact that there were also no significant group differences in family functioning (see below). Together, the results suggest that, whilst this sample of DS patients had experienced higher rates of general trauma and sexual/physical abuse, emotional abuse/neglect was not a significant factor in this group.

The above findings provide further evidence for the importance of traumatic life events in the aetiology of DS. Whilst some authors advise against attempting to obtain trauma history from patients with DS during clinical assessment (i.e. Fritzsche et al., 2013), if traumatic history is sensitively and appropriately obtained at

assessment using a standardised measure such as the TEC, this could facilitate clinicians' understanding of a patients' presentation, and also might inform which treatment options are most suitable.

#### **5.4.3. Post-traumatic symptoms**

In the subgroup of participants claiming to have been affected at least moderately by any one life event on the TEC (DS = 29, controls = 28), significantly more patients with DS met criteria for a diagnosis of PTSD than controls. This traumatised DS group scored more highly than the traumatised control group for total PTSD symptoms, and all three symptom subtypes (re-experiencing, arousal, avoidance). These findings are in accordance with previous studies indicating high rates of PTSD in patients with DS (e.g. Rosenberg et al., 2000), and are of considerable importance, both theoretically and clinically. Pathological responses to traumatic life events may play an important role in the development of DS. Unfortunately, only a subgroup of participants completed the PDS in the current study; therefore, it was not possible to include this variable in the logistic regression analyses. However, future studies might aim to explore the extent to which PTSD symptoms mediate the relationship between traumatic experience and DS diagnosis.

In the current study, PTSD symptoms were significantly correlated with several other important psychological variables in the DS group (but not in controls), including anxiety, depression, tendencies towards insecurity in relationships (IASC Abandonment Concerns) and the use of externalising behaviours to cope with emotional distress/tension (IASC Tension Reduction Activities). Whilst causality obviously cannot be assumed in these relationships, it is possible that significant trauma-related psychopathology may contribute to current mood dysfunction, relationship problems and dysfunctional externalisation of negative affect in this subgroup of patients with DS.

Some aspects of psychological dissociation (MDI Memory Disturbance, Disengagement) were positively related to PDS Avoidance symptoms. In other words, avoidance of processing trauma-related information/affect was related to symptoms of dissociative amnesia and a tendency to be detached from current

surroundings/stimuli. As dissociative symptoms are common in PTSD and the PDS Avoidance subscale includes some dissociative symptoms (e.g. “feeling distant or cut-off from the people around you”), this is not an unexpected finding and supports the assertion that dissociation and trauma-related psychopathology are interlinked.

Furthermore, PTSD symptoms (Total, Re-experiencing) were also related to ictal DS symptoms (cognitive and total respectively), indicating that elevated PTSD symptoms may be associated with worse DS symptoms, in the traumatised subgroup. Again, an interesting direction for future research might be to examine the extent to which seizure symptoms improve after patients undergo trauma-focused psychological interventions. It is possible that reductions in trauma-related psychopathology may be accompanied by reduced severity or frequency (or cessation) of DS symptoms, although this is a tentative hypothesis at present.

Given that so many patients with DS have experienced trauma/adverse life events, perceive these experiences to have had a large impact on their lives, and many suffer from PTSD symptoms, it is likely to be beneficial for these issues to be addressed within psychological interventions, at least for this subgroup. One such possibility might be the utilisation of techniques that focus on emotional processing of traumatic material, such as those with proven efficacy as PTSD interventions (e.g. prolonged exposure, EMDR) to address patients’ primary traumatic experiences (in patients who have disclosed significant trauma history/PTSD symptoms only). Further examination of the role of trauma and PTSD symptoms in patients with DS seems to be of considerable importance.

#### **5.4.4. Childhood family functioning**

Unlike several previous reports, this study did not find evidence for elevated (perceived) childhood family dysfunction in the present sample. In fact, of the five FES subscales included, only one (Conflict) came close to significance. The lack of between-groups differences on the FES subscales was not supportive of the hypotheses proposed in section 5.1. These findings are also contrary to those reported in several previous studies, summarised in Chapter 2 (section 2.2.3).

It is possible that the requirement to refer to childhood family functioning in the current study may have influenced these results. In previous research, patients may have reported difficulties occurring in family dynamics across the lifespan; therefore, rates of dysfunction may have been inflated by this. Moreover, it is possible that the lack of significant findings in the current study could be due to the stringency of the statistical methods used. For example, a between group difference on FES Conflict scores was present at the  $p < .05$  level; however, after correcting for familywise error with the Bonferroni-Hochberg procedure, this difference was no longer significant. This could represent a Type 2 error, and the use of this method may have been overly cautious.

#### **5.4.5. Anxiety and depression**

This study found evidence of elevated symptoms of anxiety and depression in patients with DS, relative to healthy controls. This is in accordance with previous research findings (e.g. Bewley et al., 2005; Hixson et al., 2006; Mökleby et al., 2002), and the hypotheses presented in section 5.1. However, mean scores for the DS group were within the non-clinical range for depression and suggestive of only mild caseness for anxiety, according to the guidelines described by Zigmond and Snaith (1983). This suggests that these symptoms were not reflective of severe emotional distress at the time of the study. The use of anti-depressant (40%) or anti-anxiety (10%) medications by a proportion of the sample (see Table 3) may have influenced the findings.

Nevertheless, HADS Anxiety scores were significantly correlated with several other variables, including PDS Total and Arousal scores. This indicates some degree of generalised hyperarousal in patients with DS, which would accord with studies indicating hypervigilance to threatening stimuli and elevated basal cortisol (i.e. Bakvis, Roelofs, et al., 2009; Bakvis, Spinhoven, & Roelofs, 2009). HADS Anxiety scores were also associated with IASC Affect Dysregulation and Abandonment Concerns, indicating that general anxiety symptoms were related to more longstanding dysregulated affect, and insecurity in close relationships.

Anxiety scores were also associated with ictal chest and abdomen symptoms during seizures, which could reflect somatic aspects of anxiety occurring during the DS (e.g.



shortness of breath, 'butterflies' in the stomach). This might suggest that the seizures are either triggered by anxiety, or a way of releasing anxiety-related distress. This is similar to the conclusions of Goldstein and Mellers (2006), although their findings related to the autonomic arousal subscale in patients with DS compared to those with ES. Future studies might explore this relationship further, by more closely examining peri-ictal somatic anxiety symptoms in patients with DS. Moreover, it might be of interest to examine possible improvements in DS symptoms after the completion of anxiety-focused treatment (e.g. cognitive behavioural therapy, anxiolytic medication).

Whilst both anxiety and depression scores independently predicted a diagnosis of DS in univariate regressions, only depression remained significant when both subscales were examined using multivariate regression. Therefore, whilst symptoms of anxiety may well be present in many patients with DS and seem to have important links to ictal symptoms, the presence of depressive symptoms is most characteristic of this group relative to healthy controls. HADS Depression scores were positively correlated with a range of other psychological variables in the DS group, including PTSD symptoms (Total, Arousal, Avoidance), borderline personality features (IASC Affect Dysregulation, Tension Reduction Activities), and dissociative amnesia (MDI Memory Disturbance). Regarding the relationships between depression and the IASC subscales, it is possible that the negative mood states associated with depression could increase the affect dysregulation scores, and it may be that this particular group cope with such negative affect by using externalising behaviours (i.e. tension reduction activities).

An important finding was that when HADS Depression scores were entered into multivariate regression analyses with other significant predictors (SDQ scores, TEC sexual abuse) and with diagnosis as the outcome variable, HADS Depression scores remained a unique predictor of DS diagnosis, alongside SDQ scores. Together, the above findings indicate that depression in patients with DS is an important factor that may contribute to the aetiology/perpetuation of DS, and may be associated with some of the other psychological difficulties observed in this group.

#### **5.4.6. Psychological and somatoform dissociative symptoms**

There were significant elevations on all subscales of the MDI, indicating that general psychological dissociation is a notable characteristic of patients diagnosed with DS. This is in accordance with much of the previous literature (see Chapter 3, section 3.3.2). Using a dimensional measure of psychological dissociation that has not previously been used in research on DS (MDI), this study provided the novel insight that a variety of different types of dissociative symptom are more common in patients with DS, compared to controls. These included depersonalisation, derealisation, disengagement, identity dissociation, memory disturbance, and emotional constriction. These various types of dissociation include examples of both ‘detachment’ and ‘compartmentalisation’, as described by E.A. Holmes and colleagues (2005).

Moreover, scores on some subscales of the MDI showed significant associations with other psychological and clinical variables in the DS group. Relationships between MDI subscales and trauma-related variables have been discussed in previous sections. Importantly, some of the MDI subscale scores were positively associated with ictal symptoms (see below section 5.4.8.). The additional finding that MDI Memory Disturbance subscale scores were positively correlated with HADS Depression scores suggested that psychologically-mediated memory problems (i.e. dissociative amnesia) were linked to general levels of emotional distress and negative mood. It is possible that the altered mood state in depression could interfere with usual cognitive processes and, therefore, lead to memory difficulties or excessive forgetting. On the other hand, depression and dissociative amnesia could be related via a third variable such as a history of trauma or PTSD avoidance symptoms.

Interestingly, the MDI Depersonalisation, Derealisation, Emotional Constriction and Identity Dissociation subscales were all positively correlated with the IASC Tension Reduction Activities subscale. Therefore, it seems that in patients with DS, a range of dissociative symptoms are associated with the use of dysfunctional externalising behaviours to cope with or express negative emotions. A tentative hypothesis might be that ongoing restriction of emotional experience and a tendency to dissociate from subjective experiences, could result in episodes of overwhelming or excessive

emotion, thereby increasing the risk of using externalising behaviours as a means to manage the emotional states. It is possible that such 'tension reduction activities' may serve the same purpose as DS; that is, providing immediate relief from intense and unmanageable emotional experience. Obviously, more research is needed to further examine these processes. The qualitative study described in Chapter 9 was designed with the aim of exploring patients' understandings of such processes.

In addition to psychological dissociation, scores on a measure of somatoform dissociation (SDQ-20) were also elevated in the patient group relative to healthy controls, and this difference remained when a seizure-related item was removed from the scores. SDQ-20 scores were also found to be one of the most important predictors of a diagnosis of DS, in combination with symptoms of depression. Moreover, mediation analyses showed that somatoform dissociation was also found to account for the predictive relationship between the presence of sexual abuse and a diagnosis of DS. These findings highlight the importance of somatoform dissociation in individuals with a history of sexual abuse who go on to develop DS. It is possible that the experience of sexual abuse increases the general tendency towards alterations in voluntary control, awareness or experience of somatic processes (somatoform dissociation); which may then increase the risk of displaying dissociative seizure-like episodes (DS), possibly along with other medically unexplained phenomena.

Some methodological and theoretical considerations should be discussed in relation to the dissociation measures. For somatoform dissociation, when some of the analyses were run excluding the seizure-related SDQ item, most of the findings remained unchanged, although not all. Many studies in the literature use measures of somatoform dissociation/somatisation which may include items about symptoms that occur during patients' seizures. Therefore, the patients will necessarily receive higher scores. It could be argued that this represents a methodological circularity; that, patients who are known to have DS will score highly on measures that assess symptoms that may occur during DS. However, given that other organic causation for the symptoms has (presumably) been excluded, those DS symptoms can be

assumed to be manifestations of somatoform dissociation/somatisation. Therefore, it could be argued that these symptoms should be included in such measures.

Either way, if investigators wish to only measure non-DS somatoform symptoms, then it is important that they select measures that either do not include items that directly relate to DS, or that such items are removed from the analyses. Furthermore, patients could be explicitly asked to complete the measures referring to symptoms that do not occur during their seizures. However, this still leaves the problem of somatoform symptoms that occur peri-ictally. For example, patients may experience sensorimotor alterations before or after their seizures, and it would be difficult to make the case that any such symptoms should be excluded from analysis. Practically, it would be very difficult to ask patients to only endorse symptoms that do not occur 'any time' around a seizure, particularly for those who experience multiple seizures in a day.

The same issues can be raised about measures of psychological dissociation. For example, the findings presented here indicate that many patients experience dissociative symptoms during their seizures. These types of symptom (i.e. mental state symptoms) are those assessed with measures of psychological dissociation (i.e. derealisation, depersonalisation), and so it could be argued that the dissociation measures are just showing that the individual experiences DS, and nothing else. However, once again, these measures have value in that they are providing more evidence that patients experience dissociative symptoms peri-ictally, and possibly more generally. Again, this provides further support to the idea that DS are dissociative phenomena.

#### **5.4.7. Borderline personality tendencies**

Several of the IASC subscales were elevated in DS patients, including Identity Impairment, Abandonment Concerns, Affect Dysregulation, and Tension Reduction Activities. These findings are suggestive of dysfunctional personality characteristics in patients with DS, including difficulties regulating emotions, the sense of self and behaviour, and insecurity in close relationships. As a number of studies have previously reported that 'Cluster B' personality traits are elevated in this group (e.g.

Direk et al., 2012; Scévola et al., 2013; Stone et al., 2004; see Chapter 2), these findings are not surprising.

When Affect Dysregulation, Abandonment Concerns and Tension Reduction Activities were entered together in a multiple regression analysis, the only significant predictor of the DS diagnosis was Affect Dysregulation. This suggests that dysregulated affect is a key feature of this patient group. However, as mentioned previously, when depression and anxiety scores were entered into the model in a second step, Affect Dysregulation was no longer a significant predictor of DS diagnosis. Furthermore, the mediation analysis confirmed that the relationship of Affective Dysregulation scores to DS diagnosis was mediated by HADS Depression scores. Therefore, once again, the importance of depressive symptoms in patients with DS is highlighted.

A number of interesting correlations were observed between some of the IASC subscales and other psychological variables, all of which have been discussed in previous sections. However, scores on the IASC subscales were not related to seizure-related variables, suggesting that these personality features may play a role in predisposing towards development of DS and associated psychological difficulties, but not necessarily in causing or worsening specific symptoms of the disorder, once initially triggered.

#### **5.4.8. Ictal symptoms**

The most commonly reported ictal symptoms were cognitive, mental state and autonomic arousal (AA) symptoms. Whilst the current study did not include a seizure-comparison group, the mean number of AA symptoms reported by the DS group in the present study was closer to the mean of the DS group (2.64) from the Goldstein and Mellers (2006) study, rather than the mean of the ES group in that study (1.63). These findings indicate that symptoms of physiological arousal, resembling the somatic manifestations of anxiety, are a common experience during DS. These results support the proposition that elevated levels of emotional arousal/anxiety/distress may serve as an important factor in triggering DS (Baslet, 2011; Goldstein & Mellers, 2006).

The 'mental state' (MS) symptom type was also commonly reported in the current sample of patients. The MS category primarily consists of symptoms resembling those of depersonalisation and derealisation, in addition to one item about faintness and dizziness. The high rates of ictal MS symptoms observed in this study suggest that symptoms of dissociation, especially of the 'detachment' type are commonly experienced by patients during or around the time of their attacks. Furthermore, scores on the MDI Derealisation and Depersonalisation subscales were significantly correlated with ictal MS symptoms, strengthening this proposition. It could be that patients who experience more MS symptoms during their attacks also experience higher levels of depersonalisation and derealisation more generally (i.e. in daily life). On the other hand, the elevated MDI Depersonalisation and Derealisation scores could be directly reflecting higher levels of such symptoms during the seizures. Nevertheless, it is clear that symptoms of the 'detachment' type of dissociation are experienced by a significant proportion of patients with DS, either during their seizures, generally in daily life, or both.

Other common ictal symptoms reported in the current sample were those belonging to the 'cognitive' category. This category includes items that reflect cognitive manifestations of anxiety (e.g. wanting to escape a situation, embarrassment, fears of death, paralysis, or losing consciousness). It is possible that these anxiety-related cognitions are possibly the result of the severe and probably rather frightening loss of control and voluntary physical functioning that patients experience during a DS. It would be interesting to examine these symptoms in more depth, particularly during the peri-ictal period, in order to examine the progression of such experiences and how they relate to the onset and course of the attacks.

A potentially valuable direction for future research, therefore, seems to be the further examination of patients' peri-ictal subjective experiences. For example, do patients experience a specific alteration in emotional experience, akin to 'depersonalisation' or 'emotional constriction' during their seizures? It might be beneficial to develop measures that could quantify such experiences in more detail. Such a questionnaire might involve a Likert-scale for a range of different emotions, such as fear,

contentment, calmness, anger, rage, and so on. Alternatively, another possibility could be to provide patients with a measure of state dissociative experiences (e.g. the State Scale of Dissociation; Kruger & Mace, 2002), and request that patients complete the questionnaire immediately on recovery from seizures, with reference to their experiences during the seizures. Furthermore, focused qualitative techniques could be used to explore patients' emotional experiences, or lack thereof, during DS. An obvious limitation of this approach would be the necessary exclusion of patients who report loss of awareness during their attacks.

#### **5.4.9. Strengths and limitations**

A key strength of this study was the examination of a wide number of potentially important aetiological variables in one sample of patients with DS, and the inclusion of a matched control group. This allowed an examination of how patients' responses on these measures differed from those of healthy individuals, but also analyses of the interactions and relative importance of these variables within- and between-groups (e.g. exploratory analyses). The two groups included in the study were well-matched on several variables that could have possibly influenced the findings obtained, such as gender, age and SES. Moreover, clear definitions of each variable were provided, with standardised measures used for all. This also permitted comparison of patients' scores to normative standards, where relevant. Furthermore, some of the measures had not been used in previous studies of patients with DS, such as the IASC and MDI, and so provided novel tests of some of the hypotheses set out in section 5.1. The study has provided support for, and extended previous research findings pertaining to psychosocial factors in patients with DS, whilst also presenting novel findings that highlight possible avenues for future research.

More specifically, the assessment of ictal symptoms provided a means by which relationships between seizure symptoms and psychosocial variables could be explored. In addition, the use of the TEC to examine adverse life experiences provided information regarding a variety of life events in patients with DS, but also patients' perceptions of the impact of these events. This was examined in more detail with the PDS in traumatised patients, again, representing a thorough assessment of the effects of adverse life events in this group. Many previous studies have not used

standardised measures or assessed the subjective impact of life events in patients with DS.

Another strength of the study was the inclusion of a multidimensional measure of dissociation (rather than measures such as the Dissociative Experiences Scale), allowing an examination of different types of dissociative symptom and how these related to seizure symptoms, for example. Moreover, measuring both somatoform dissociation and psychological dissociation in the same sample provided insight into the relative importance of these types of experience. The use of the IASC also provided a structured assessment of patients' personality functioning, particularly in relation to borderline features. The findings on this measure not only indicate the possible aetiological contribution of such characteristics but also suggest implications for treatment-related processes (e.g. the presence of insecurity in relating to others, considerable affect dysregulation).

There are some limitations that should be acknowledged, however. The use of self-report measures could be seen as a limitation when assessing some variables, due to possible biases arising from self-presentation concerns or demand characteristics. It was also not possible to validate trauma reports and so the data was dependent on participant recollection and willingness to disclose. Additional limitations in the current study pertain to statistical techniques. Several of the variables were not normally distributed; therefore, non-parametric techniques were used in some analyses. Non-parametric techniques are generally thought to have less power to detect effects (D. Howell, 1997); therefore, the use of such tests may have inflated Type 2 error rates (e.g. the unexpected negative findings on the FES). In contrast, it could be argued that type I error rates were inflated by the use of multiple testing in some analyses. However, the use of the Bonferroni-Hochberg (Hochberg, 1988) correction for multiple testing of subscales on the various measures is a rather conservative approach. It is known that the Bonferroni correction is a very stringent method of controlling familywise error (i.e. Shaffer, 1995). The Hochberg adaptation to this method maintains good control of familywise error whilst improving power (Hochberg, 1988; Wright, 1992).



Due to the large number of tests carried out in the exploratory analyses, rather than utilising corrected p-values, a more stringent alpha level was selected ( $p < .01$ ) by which to assess significance. Again, caution should be exercised in interpretation of significant effects found in these exploratory analyses. The hypotheses generated from these analyses should be tested using more stringent techniques in further studies. An additional point regarding the multivariate regression analyses is that, whilst the models were significant, in some cases the rate of successful classification of group status was rather limited (e.g. approximately 70% accuracy), suggesting that some proportion of the variance was not explained by the variables entered.

#### **5.4.10. Summary and conclusions**

The findings presented in this chapter indicate high levels of adverse life events and considerable psychological impact of these experiences in patients with DS. The importance of sexual abuse was highlighted for differentiating patients with DS from healthy controls, and together these findings suggest the possible utility of assessing traumatic background early in the clinical management of such patients. The findings also provide further evidence that a range of psychopathological symptoms are elevated in patients with DS, relative to healthy control participants. These include anxiety, depression, PTSD symptoms, and some borderline personality features. Moreover, elevated psychological and somatoform dissociation was also a feature of the patient sample. Some of these phenomena were inter-related and some positively associated with seizure symptoms.

Of the variables measured, somatoform dissociation and depression seem to be important variables characterising patients with DS relative to healthy controls. This study also suggested that these variables might mediate the relationship between sexual abuse and DS diagnosis. Again, clinical applications involve the possibility of using a formal measure of somatoform dissociation and depressive symptomology at some stage during assessment of patients with DS, in addition to explicitly addressing such symptoms within psychological interventions for the disorder.

## **Chapter 6. Preconscious facial expression processing in patients diagnosed with dissociative seizures**

### **6.1. Introduction**

#### **6.1.1. Background**

The current chapter describes an experimental study of preconscious processing of emotional facial expressions in patients diagnosed with DS, in comparison to healthy control participants. In Chapter 3 (section 3.6) it was proposed that aberrant responses to emotional stimuli, possibly occurring without patients' full awareness, might contribute to triggering DS. Furthermore, it was suggested that a general tendency towards altered preconscious emotional processing might be a predispositional characteristic that increases the risk of developing the disorder. It is possible that preconscious attentional biases towards affective stimuli, particularly those of a negative or distressing nature, could increase overall levels of emotional arousal and stress in this patient group. Such a bias might increase the likelihood of seizure reoccurrence, by elevating arousal or distress to unacceptable levels. Therefore, investigating the ways in which patients with DS process emotional stimuli at a preconscious level is an important avenue for research on DS.

In discussing preconscious processing, it is important to define what is meant by this term. Whilst widely accepted definitions of consciousness are rather elusive (Devinsky, 1997), Pinker (1997) proposed three distinct aspects of this enigmatic phenomenon. The term 'sentience' generally refers to phenomenological experience, that is, one's own individual, subjective and private existence. On the other hand, 'access to information' is the aspect of consciousness involving awareness of mental processes or the external world. Finally, 'self-knowledge' involves accessing information pertaining to oneself, and might also be referred to as self-awareness. Since the 1980s, psychologists have started to pay more attention to the 'access level' of consciousness using experimental measures, including examining the processes that occur below the threshold of conscious awareness (Merikle, 2007). Findings pertaining to automatic and unconscious cognitive processes could be viewed as one

of the major advances in the scientific understanding of consciousness in the last half century (Frith & Rees, 2007).

Regarding the 'access' approach to consciousness, 'conscious' processes refer to content that is currently accessible (i.e. available to introspection, that which can be reported); the preconscious refers to information and material that is not currently within awareness, but of which we can become aware if attention is directed at it. Finally, unconscious processes are presumably those which are not accessible at all, despite the allocation of attention to them. Responses to sensory information that occur relatively automatically and below the level of conscious awareness can be thought of as 'zombie modes', allowing quick but rather stereotyped responses to environmental stimuli (Crick & Koch, 2007). Tamietto & de Gelder (2010) and Dehaene et al. (2006) discuss some of the various terms used to describe 'non-conscious' processing of stimuli. These terms include: implicit, subliminal, preattentive, automatic and preconscious. Whilst these terms are distinguishable on the basis of subtle differences, they all refer to instances in which stimuli are processed without explicit awareness or attention.

From an evolutionary perspective, it is likely that stimuli that had significance to the survival goals of our ancestors are more likely to be processed in an automatic or non-conscious fashion. Öhman and colleagues (2000) argue that facial expressions, particularly those relating to threat (e.g. anger, fear), are innately salient stimuli for human beings because they confer crucial social signals that have considerable implications for survival. Furthermore, there is evidence that such stimuli capture attention and can provoke autonomic responses without awareness (Eastwood, Smilek, & Merikle, 2003; Esteves, Dimberg, & Öhman, 1994; Öhman, 2002; Öhman, Flykt, & Esteves, 2001; Pessoa et al., 2005; Vuilleumier, 2002; Vuilleumier & Schwartz, 2001).

#### **6.1.2. Previous research using modified Stroop paradigms**

Since the pioneering work of Robert Zajonc (1980), there has been an enormous amount of research on what is now known as 'automatic affective processing'. One paradigm that has commonly been used to measure automatic or preconscious

emotional processing is the 'emotional Stroop' test (Williams, Mathews, & MacLeod, 1996). This test is a modification of the classic Stroop test (Stroop, 1935). In the emotional variant, the to-be-read words are selected to be in some way emotionally significant. These might include positively or negatively valenced words (e.g. hate, fun, happy, misery), with neutral words included as a control condition. The dependent variable is typically reaction time, although error rates can also be examined. Increases in reaction time for a given stimulus condition are interpreted as being due to increased attentional allocation to such stimuli, that is, hypervigilance or an attentional bias. In contrast, shorter reaction times are generally interpreted as evidence of automatic avoidance of the stimuli. More errors on a particular condition would indicate greater cognitive interference by the stimuli in that condition.

The emotional Stroop task has been used extensively in studies with a wide variety of clinical populations, including the anxiety disorders (e.g. generalised anxiety, PTSD, panic), major depression, and somatoform disorders. Often, in such studies, rather than including words with general emotional content, words with some relevance to the condition under investigation are selected. A further modification of the task is the use of emotionally-significant images, rather than words (e.g. Lee et al., 2009). Again, investigators have used pictorial versions of the test to study attentional biases in a wide range of psychological disorders (e.g. Harrison et al., 2010; Hermans et al., 2006). Within such tasks, many investigators present the emotional stimuli subliminally; most commonly using very short stimulus durations (milliseconds), and/or using masking stimuli. There is debate about the exact duration of stimulus presentation that precludes conscious awareness, but it appears to be in the range of approximately 1-33 milliseconds; although individual differences are apparent (Esteves & Öhman, 1993; Maxwell & Davidson, 2004).

Considerable research evidence has accumulated regarding automatic affective processing in clinical populations that are of relevance to DS. There is a good evidence base for the proposition that symptoms of anxiety and anxiety disorders are related to increased allocation of attention to threat-related stimuli (e.g. Bar-Haim et al., 2007; Lim & Kim, 2005; Mogg & Bradley, 1999; 2002; B.P. Bradley et al., 1999; Vuilluemier,

2002), although other patterns have also been reported (e.g. Putman et al., 2004). Nevertheless, there is considerable evidence of attentional bias towards threatening/trauma-related stimuli in patients diagnosed with PTSD, as measured using emotional Stroop tasks, among others (Buckley, Blanchard, & Neill, 2000; R.J. McNally, English, & Lipke, 1993).

Individuals with depression also show altered attentional allocation to affective stimuli. A meta-analysis of studies investigating attentional biases to negative stimuli in depressed individuals (including emotional Stroop and dot probe tests), provided evidence for disproportionate allocation of attention to negative stimuli (Peckham, McHugh, & Otto, 2010). It has been proposed that these biases towards allocating attention to negative stimuli may contribute to the core symptoms of depression, such as negative mood and anhedonia (Phillips et al., 2003).

Individuals diagnosed with borderline personality disorder (BPD) also seem to exhibit attentional biases towards negative and BPD schema-related stimuli (Arntz, Appels, & Sieswerda, 2000; Sieswerda et al., 2007). In one of these studies, biases towards negative schema-related stimuli were significantly predicted by the severity of BPD symptoms (anxiety subtype), and reported childhood sexual and physical trauma (Sieswerda et al., 2007), suggesting the importance of examining the ways in which attentional biases relate to other characteristics of the clinical group being studied. Nevertheless, there have also been negative findings in the literature, in which biases have not been observed in patients with BPD (Wingenfeld et al., 2008). In a fairly recent review, Baer et al (2012) concluded that, in general, attentional biases for negative stimuli are apparent in BPD, but that these are not necessarily specific to 'BPD-related' themes (i.e. abandonment, vulnerability).

A small number of investigators have also studied these processes in patients with dissociative diagnoses (Dorahy, Middleton, & Irwin, 2005; Hermans et al., 2006). Hermans and colleagues (2006), for example, utilised an emotional Stroop paradigm to assess DID patients' responses to subliminally presented angry faces. When the DID patients were tested in a 'trauma-avoidant' identity state, they showed significantly reduced reaction times to angry faces, compared to controls. This was

interpreted as an adaptive avoidant response. However, this study utilised a very small sample size, with only female student control participants included. Moreover, no statistical comparisons were made on age or years of education, and no information was provided on psychiatric comorbidity or medications in the clinical group. These weaknesses limit the interpretability of the results and highlight the importance of matching for, or controlling possible confounding variables.

A tendency towards somatisation may also be associated with altered automatic processing of some types of stimuli. Roelofs et al. (2002) carried out a meta-analysis of studies using the modified Stroop paradigm with patients diagnosed with chronic pain, and found evidence for attentional biases towards pain-related stimuli in that group. Moreover, individuals diagnosed with somatoform disorder have also been reported to show increased interference to physical threat words compared to neutral words on an emotional Stroop task (Lim & Kim, 2005); however, performance on such tasks seems to be influenced by tendencies towards alexithymia (Mueller, Alpers, & Reim, 2006) and emotion suppression in these patients (Wingenfeld et al., 2011).

As discussed in Chapter 3 (section 3.5.2), a landmark study was carried out in the Netherlands by Bakvis, Roelofs, and colleagues (2009), in which patients with DS and healthy control participants were tested with an emotional Stroop paradigm including negative (anger), neutral, and positive (happy) facial expressions. The details of this study were provided previously, but in brief, patients with DS displayed significantly greater attentional bias towards angry faces relative to the control group (within a baseline condition, but not after stress-induction). The attentional bias scores were positively associated with self-reported history of sexual abuse and elevated basal cortisol levels. This study suggested that angry facial expressions caused a disproportionate preconscious reallocation of attention in the DS patients, indicating that these stimuli were particularly salient for this group.

Bakvis, Roelofs, and colleagues' (2009) study had a number of strengths, including the exclusion of patients taking medications, the inclusion of only patients diagnosed with video-EEG, the assessment of trauma history and physiological measures of stress

reactivity (e.g. salivary cortisol), and the use of backward-masking and an awareness check to ensure that stimuli were processed subliminally. This was an important piece of research, as it was the first experimental study in this patient group. Therefore, it was a novel approach to investigating psychological processes in DS. It should be noted, however, that the study also had several important limitations. These included a small (19 patients, 20 controls) and potentially biased/unrepresentative sample, due to the likely exclusion of large numbers of patients due to medication or lack of video-EEG evidence for diagnosis. Moreover, whilst information was provided regarding additional diagnoses in the clinical group, current symptoms of anxiety and depression, for example, were not measured. It is well known that emotional processing is influenced by such symptoms; therefore, failing to consider the influence of these is a significant weakness. Furthermore, there was no indication as to the severity or extent of the reported traumatic life events (e.g. sexual abuse).

A number of studies have indicated the possible presence of subtle neurocognitive deficits, non-specific neurological anomalies, and a history of minor head injury in patients diagnosed with DS (see Chapter 2, sections 2.3.6, 2.1.3, and 2.1.2 respectively). Despite this, Bakvis, Roelofs, et al. (2009) did not report data on cognitive abilities, such as general intellectual functioning or face processing. It, therefore, remains a possibility that differences on the emotional Stroop test may have been influenced by such variables (although a classic Stroop test was included, on which patients performed similarly to controls).

### **6.1.3. Rationale, aims and hypotheses**

The study described in the current chapter was conducted with several aims. One important aim was to attempt to replicate the findings of Bakvis, Roelofs, et al. (2009) in a larger and more representative sample of patients with DS recruited in London, UK. The aim was to recruit a sample of 40 patients with DS and compare their performance to the same number of healthy controls, in order to provide a higher level of statistical power than the previous study. Moreover, the study also set out to control for the possible influence of anxiety and depression, in order to provide a

more DS-specific assessment of performance on the experimental test. Whilst anxiety and depression are commonly elevated in DS, they are also not uncommon in the general population, individuals with ES, and patients with other psychiatric diagnoses.

In order to achieve the stated aims, an emotional Stroop task was devised that was as similar as possible to that described by Bakvis, Roelofs, et al (2009). The test was administered to a group of patients diagnosed with DS and a group of healthy control participants. Anxiety and depression scores (and YoE) were covariates in the research design. On the basis of the literature (reviewed above), the following hypothesis was tested:

After controlling for symptoms of depression and anxiety, patients with DS were predicted to show elevated attentional biases towards emotional faces, compared to the healthy control group. This difference was expected to be most apparent for angry facial expressions.

Moreover, exploratory correlational analyses were carried out with the objective of exploring whether important psychosocial factors (those found to differ between-groups in Chapter 5 and, therefore, assumed to be aetiologically relevant) were related to the attentional bias (AB) scores for happy or angry faces. Relationships between AB scores and the following psychosocial variables were explored: psychological and somatoform dissociation, trauma history, anxiety, depression, post-traumatic symptoms, and borderline personality characteristics (abandonment concerns, identity impairment, affect dysregulation, tension reduction activities).

In addition, within the DS group, possible relationships between attentional bias scores and seizure-related variables (e.g. DS frequency, duration of DS disorder, ictal symptoms) were also explored. These variables were included in the analysis because, if preconscious affective processing biases are in some way involved in predisposing towards DS or triggering individual attacks, such relationships might be expected.



## **6.2. Methodology**

The task was included in an initial pilot study, described in Chapter 4 (Section 4.1). No modifications were made to the procedures as a result of this pilot work, as the task was found to be generally acceptable to participants.

### **6.2.1. Participants**

Details of recruitment procedures and inclusion criteria for this study were described in Chapter 4 (sections 4.2.1. and 4.2.2). The final sample for this study included 43 control participants and 38 patients diagnosed with DS.

### **6.2.2. Materials**

#### ***Experimental materials***

The facial stimuli used in the experiment were pictures of models displaying angry, happy or neutral facial expressions taken from the standardised 'Pictures of Facial Affect' set (Ekman and Friesen, 1976). These stimuli have been standardised in normative samples cross-culturally. Expressions of happiness, anger and neutrality were selected because they would provide examples of both positive and negative emotions, and a control condition. These were also the emotions used in the study reported by Bakvis, Roelofs, et al. (2009).

Pictures of five male and five female models were utilised in order to minimise any gender effects within the stimuli. This yielded a total of 30 stimuli. The faces were cropped and superimposed on a black background, in order to standardise them and remove any potentially distracting features (e.g. hair, ears). The grey-scale images of the faces were coloured in a transparent shade of red, yellow or green, which allowed the facial expressions and features to remain clearly visible. Examples of these stimuli and a list of all the stimuli used can be found in Appendix 12. The masking stimuli were neutral patterns (see Appendix 12), consisting of several high-contrast concentric ovals in red, green or yellow (identical shades to those used for the facial stimuli), presented on a black background. These stimuli were constructed so that the outer edge would resemble the shape of the cropped faces, and so that they would completely cover the same area of the screen as the facial stimuli.

### ***Neuropsychological measures***

*Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999)*

The two subscale form of this test was administered to briefly assess current intellectual functioning (Matrix Reasoning, Vocabulary). The reason for inclusion of this test was to ensure that the groups were matched on general cognitive abilities, as these may have influenced performance on the experimental task.

The Matrix Reasoning subtest requires participants to select an item from an array, in order to complete a pattern or rule presented within a matrix. The Vocabulary subtest involves the examiner presenting single words orally and visually, with the participant being required to provide a definition of the meaning of each word in turn. The two tests are associated with Performance and Verbal IQ respectively (PIQ, VIQ), and yield T-scores based on the normative data provided in the manual. The combined T-scores from the subtests are used to calculate an overall Full Scale IQ score (FSIQ). This test was selected as a time-efficient means of obtaining a good estimate of overall cognitive functioning, for participant matching purposes. The time constraints of the testing procedures would not have allowed for the full Wechsler Adult Intelligence Scale (Wechsler, 2008) to have been administered.

*Stroop Test (Golden & Freshwater, 2002)*

A standardised version of the Stroop test was administered to assess general executive functioning (response inhibition/attention/processing speed). Given that the experimental task places some degree of load on executive skills, it was thought necessary to ensure that the groups were well-matched on this variable.

The standard Stroop test includes three conditions. The first condition (Word) is a basic reading test, in which respondents are asked to read a list of colour words (red, yellow green) all printed in the same colour (black), as quickly as possible for 45 seconds. The number of correctly read words is then recorded. The second condition (Colour) requires respondents to say aloud the colour of a series of stimuli (xxxx) printed in red, yellow, or green. The number of correctly labelled items is recorded. The final condition (Colour-Word), requires respondents to name the

colour of the ink that each colour word is printed in, whilst ignoring the actual word. So, for example, respondents may have to say 'green' when the word 'red' is printed in green ink. This condition requires respondents to suppress the automatic tendency to verbalise the written word, whilst simultaneously selecting and verbalising the correct colour of the ink for each item. The total number correctly stated in 45 seconds is once again recorded.

On the basis of age, educational level, colour and word condition scores, predicted values for the 'colour-word' condition scores can be obtained from the normative data provided in the test manual. It is then possible to calculate 'interference' scores by subtracting the actual colour-word score from the predicted colour-word score. Raw 'interference' scores are then used to calculate a T-score, for reference to the normative sample. According to Jensen (1965) and Golden (1975), the Stroop test shows good test-retest reliability in the .70-.90 range (as cited by Golden & Freshwater, 2002).

#### *Benton Facial Recognition Test (short-form; Benton et al., 1983)*

This test requires participants to match faces presented simultaneously, on the basis of the physical features of the individual (identity). This provides a basic measure of general perceptual processing of facial stimuli. The test was included to ensure that participants in both groups had facial processing abilities that were within the normal range, due to the presentation of facial stimuli in the experimental task. The short-form test was used due to time constraints. However, the short-form scores are easily converted to the long-form equivalent, which can be adjusted with respect to a participant's age and years of education. Scores below 29 are interpreted as impaired facial processing, with scores of 40 or below indicating borderline impairment.

### **6.2.3. Design**

This was a mixed between- and within-subjects design with two independent variables: one between-subjects variable with two levels (Group: DS, control) and one within-subjects variable. The within-subjects variable had three levels for absolute reaction times (RTs; facial expression type: happy, angry, neutral) and two

levels for attentional bias (AB) scores (facial expression type: happy, angry). The dependent variables were absolute reaction times (RTs) or AB scores (see section 6.2.5).

There were 30 stimuli for each facial expression type (happy, angry, neutral), with ten of each colour (red, yellow, green). The total number of trials was 90, with these being presented in a different pseudo-randomised order for each participant. No more than two consecutive presentations of stimuli from the same condition (facial expressions type) or of the same colour were permitted, in order to avoid the possibility of order effects within stimulus presentation.

#### **6.2.4. Procedure**

Before commencing the task, participants were presented with standardised instructions on the screen, in white font (size 16-18; Tahoma style) on a black background. These instructions can be found in Appendix 13. The experimenter read each screen aloud for participants and checked for comprehension before moving onto the next screen. Any questions that arose during this process were answered at the time.

The task began with nine practice trials. Practice trials consisted of a 750 millisecond (msec) presentation of a fixation cross (central white cross on black background), followed by the neutral pattern stimuli in red, yellow or green on each trial (a total of three of each colour in a random order). Participants were requested to say aloud the colour of the pattern as quickly as possible. On successful registration with the voice key device, the pattern disappeared from the screen. If voice key registration failed, the stimulus remained on the screen and participants were asked to respond again. The inter-trial-interval (ITI) was fixed at two seconds, during which the screen was blank.

Following completion of the practice items, and after any further questions had been answered, participants were prompted to begin the main task by pressing the space key. Each trial started with presentation of a white fixation cross (750 msecs), followed by a facial stimulus, presented for 17 milliseconds in red, yellow or green.

The face was then masked immediately by a neutral pattern stimulus in the corresponding colour. Participants were required to name aloud the colour of the masking stimulus as quickly as possible. The masking stimulus remained on the screen until participants' verbal response was registered by the voice key. Presentation of the facial stimuli was set at 17 milliseconds because this was the minimum refresh rate for the laptop-integrated monitor used in the experiment. This was four milliseconds longer than the duration of presentation used by Bakvis, Roelofs, et al. (2009); however, it is within the range of 0-33 milliseconds suggested to be the duration at which conscious awareness is precluded (see section 6.1).

The experimenter coded the participants' responses for each trial. Responses were coded by the colour verbalised (i.e. red, yellow, green) or as a mistrial (x) for instances in which the voice key failed or in which the voice key was erroneously triggered by random noise. Coding took up to two seconds per trial, during which the screen was blank. The screen remained blank for a further two seconds after the trial had been coded. This constituted a variable ITI of between approximately two and four seconds, in which participants viewed a continuous blank screen.

In order to assess whether the facial stimuli had been consciously perceived, at the end of the task participants were asked whether they had been aware of seeing any additional stimuli on the screen, and responses were recorded. If the response was affirmative, participants were asked what they had seen. An objective awareness check was also carried out, after the emotional Stroop test (and two other experimental tests, see Chapters 7 and 8) had been completed. This involved a forced-choice task in which 30 trials (identical to the experimental trials) were presented. Participants were explicitly required to select which facial expression they had seen from three choices (happiness, anger, neutral), using the number keys on the keyboard. There were 10 of each facial expression, presented in a pseudo-randomised order. This test was used as an objective measure of the degree to which the faces were available to participants' conscious awareness.

### **6.2.5. Data analysis**

General statistical procedures and considerations were described in Chapter 4 (section 4.5). Only those aspects of the data analyses specific to the current study are outlined below.

For the awareness check, correct responses were defined as correct selection of the emotion of the subliminally-presented facial expression. Percentage correct scores (0-100%) were calculated for each participant, by condition (happy, angry, neutral). A mixed factorial ANCOVA was used to examine the effect of group (between-subjects; DS, control) and facial expression (within-subjects; happy, angry, neutral) on percentage correct scores, whilst covarying for years of education (YoE). Furthermore, a binomial test was used to determine whether the percentage correct scores in each group were significantly different from chance performance.

During data cleaning, RTs for correct responses on the emotional Stroop test were checked twice for accuracy of data input; any inaccuracies were corrected. RTs were then removed if they fell below 150 or above 1500 milliseconds; as such RTs are suggestive of technical errors with voice-key registration or participant variables such as distraction, impulsivity or other processes that invalidate the responses. Furthermore, individual RTs were considered outliers and removed if they fell above or below 2.5 standard deviations from the group mean for each condition (happy, angry, neutral). New overall group means for each condition were then calculated and subjected to analysis. These two steps were also used by Bakvis, Roelofs, et al. (2009). In addition, error rates were calculated by condition.

The absolute RTs obtained on the emotional Stroop test were examined with a mixed factorial ANCOVA, with group (DS, control) as the between-groups factor and facial expression (happy, angry, neutral) as the within-groups factor. YoE and anxiety and depression scores (HADS) were entered as covariates. This analysis was conducted to assess possible group differences in speed of responding for all facial expression types, including neutral expressions. The rationale for conducting this analysis was to examine whether or not there were differences between groups on overall

reaction times within the test. Particularly, this allowed an assessment of whether the DS sample exhibited any general reductions in processing speed (as might be expected due to possible medication effects).

Moreover, AB scores were calculated by subtracting the mean RT for the neutral condition from the mean RT for each expression (happy or angry), for each participant. This method provides a gauge of how fast or slow RTs were for each emotional expression (happy, angry), relative to the neutral expressions, for each individual. These scores were assessed with ANCOVA as described above, with AB scores as the DV and with expression (anger, happiness) as the within-groups factor. Calculation of AB scores followed the method described by Bakvis, Roelofs, et al. (2009).

The exploratory correlational analyses consisted of bivariate correlations between AB scores and scores on the MDI (Depersonalisation, Derealisation, Disengagement, Emotional Constriction, Identity Dissociation, Memory Disturbance subscales), SDQ-20, TEC (total, mean impact, physical and sexual abuse scores), HADS (Anxiety, Depression subscales), PDS (Total, Arousal, Avoidance, Re-experiencing subscales), and the IASC (Abandonment Concerns, Identity Impairment, Affect Dysregulation, Tension Reduction Activities subscales). Furthermore, possible relationships between AB scores and patient characteristics were also examined in the DS group, including seizure frequency, duration of DS disorder, medication use (AEDs, antidepressants), and ictal symptoms. These were two-tailed correlations and a stringent alpha level of  $p < .01$  was adopted to test for significance.

## **6.3. Results**

### **6.3.1. Participant characteristics**

The demographic characteristics of the control sample ( $n=43$ ) included in this experiment were as described in Chapter 5 (see Table 2). Thirty-eight patients with DS completed the current experiment. Table 28 provides the demographic

characteristics of this sample and statistical values for comparison with the control group.

There were no significant between-group differences in age, or the proportion of female, right-handed, white participants or smokers. Furthermore, the proportion of participants reporting 'higher managerial, administrative or professional occupations' (NSSEC) did not differ between groups. There was an almost significant difference in years of education between-groups; therefore, this variable was included in the analysis of the experimental data. Despite this, the proportion of participants having achieved further or higher education qualifications did not vary by group. Being in a long-term relationship (married/cohabiting) was significantly more common in the DS sample relative to controls.

Furthermore, significantly more participants in the DS group were taking prescribed medications and had general medical diagnoses, compared to the control group. Thirteen patients with DS (34%) were taking AEDs, most commonly sodium valproate (n = 3), pregabalin (n = 4), and carbamazepine (n = 3). Sixteen patients reported taking antidepressant medications, most commonly SSRIs such as fluoxetine (n = 6) and citalopram (n = 4).

For the DS group, the mean current seizure frequency was 20.4 per month (range 0-274, standard deviation = 48.1), with the median being 4.33 (interquartile range = 14.6). The mean length of time since seizure onset was 90.9 months (range 9-432 months, standard deviation = 88.4 months), with a median of 54 months (i.e. 4.5 years; interquartile range = 96.8 months).

### **6.3.2. Neuropsychological measures**

A summary of the findings for the neuropsychological measures can be found in Table 29. As some measures were not completed by all participants, the number of participants who completed each test is also included in this table.



**Table 28. Characteristics of participants completing the preconscious facial expression processing experiment**

|                               | <b>Dissociative seizures<br/>(n = 38)</b>                        | <b>Test statistics<br/>(comparison with<br/>control group)</b> |
|-------------------------------|------------------------------------------------------------------|----------------------------------------------------------------|
| Age in years                  |                                                                  |                                                                |
| Mean (SD)                     | 39.2 (12.6)                                                      | U (81) = 720, p = .358                                         |
| Median (IQR)                  | 4.5 (22.5)                                                       |                                                                |
| Gender                        | Male = 8 (21%)<br>Female = 30 (79%)                              | $X^2$ (1, n = 81) = .076, p = .782                             |
| Handedness                    | Right = 29 (76%)<br>Left = 6 (16%)<br>Ambidextrous = 3 (8%)      | $X^2$ (1, n = 81) = 2.05, p = .152                             |
| Ethnicity                     | White = 30 (79%)<br>Non-white = 8 (21%)                          | $X^2$ (1, n = 81) = 1.9, p = .168                              |
| YoE                           |                                                                  |                                                                |
| Mean (SD)                     | 12.9 (2.6)                                                       | U (81) = 616, p = .054                                         |
| Median (IQR)                  | 13 (3.25)                                                        |                                                                |
| Qualifications                | GCSEs or none = 14 (37%)<br>Further / higher = 24 (63%)          | $X^2$ (1, n = 81) = .251, p = .113                             |
| Marital status                | Currently single = 22 (58%)<br>Long-term relationship = 16 (42%) | $X^2$ (1, n = 81) = 4.24, p = .04                              |
| Socio-economic status (NSSEC) | 1 = 17 (45%)<br>2,3,4 or 5 = 21 (55%)                            | $X^2$ (1, n = 81) = .068, p = .794                             |
| Medication use                | Yes = 27 (71.1%)<br>No = 11 (28.9%)                              | $X^2$ (1, n = 81) = 18.6, p < .001                             |
| Medical diagnosis present     | Yes = 23 (60.5%)<br>No = 15 (39%)                                | $X^2$ (1, n = 81) = 19.04, p < .001                            |
| Smoking                       | Yes = 11 (29%)<br>No = 27 (71%)                                  | $X^2$ (1, n = 81) = .697, p = .404                             |

SD = standard deviation

IQR = interquartile range

YoE: years of full-time education (or equivalent)

NSSEC: National Statistics Socio-economic Classification system

1 = Higher managerial, administrative and professional occupations

2 = Intermediate occupations

3 = Small employers and own account workers

4 = Lower supervisory and technical occupations

5 = Semi-routine and routine occupations

**Table 29. Neuropsychological findings (emotional Stroop task)**

|                                       | <b>DS</b>    | <b>Control</b> | <b>Test statistics</b>   |
|---------------------------------------|--------------|----------------|--------------------------|
| <b>WASI</b>                           | n = 38       | n = 43         |                          |
| FSIQ (Mean, SD)                       | 104.1 (14.7) | 108.1 (13.1)   | t (79) = 1.29, p = .198  |
| Vocabulary T scores (Mean, SD)        | 51.8 (11.3)  | 55.2 (9.8)     | t (79) = 1.46, p = .148  |
| Matrix reasoning T scores             |              |                |                          |
| Mean (SD)                             | 52.5 (8.9)   | 53.7 (9.5)     |                          |
| Median (IQR)                          | 54.5 (10)    | 56 (15)        | U (81) = 730, p = .410   |
| <b>Benton Facial Recognition Test</b> | n = 37       | n = 43         |                          |
| Mean (SD)                             | 47.2 (4.4)   | 48.9 (3.5)     |                          |
| Median (SD)                           | 49 (7)       | 49 (5)         | U (80) = 631, p = .109   |
| <b>Stroop test</b>                    | n = 37       | n = 43         |                          |
| Mean (SD)                             | 52.8 (8.3)   | 51.5 (9.01)    | t (78) = -.656, p = .514 |

SD = standard deviation; WASI = Wechsler Abbreviated Scale of Intelligence; FSIQ = Full-Scale IQ;  
DS = dissociative seizures

On the WASI, there were no significant between-group differences in full-scale IQ, or on the Vocabulary or Matrix Reasoning subscales. All participants scored over 70 for WASI full-scale IQ. In addition, the control and DS groups performed comparably on the standardised version of the Stroop test, with no statistical difference observed in interference scores. The mean scores for both groups were in the normal range. Performance on the BFRT did not differ statistically between the DS and control groups. Moreover, the mean and median values for each group were in the normal range. However, a minority of the participants (DS = 4; control = 1) had scores in the borderline range (scores of < 40) on the BFRT.

### 6.3.3. Awareness check

After covarying for YoE, there was no overall effect of group on the percentage of correct responses on the awareness test ( $F(1, 78) = 3.275, p = .074$ ). There was no overall effect of expression ( $F(2, 78) = 1.341, p = .265$ ), and no interaction of expression by group ( $F(2, 78) = .402, p = .669$ ). The same pattern was observed when HADS Anxiety and Depression scores were added as covariates. Binomial tests were carried out to determine whether performance differed from chance in each group, with a stringent alpha level of  $p < .01$  as the criterion for significance. The results suggested that the mean percentage correct scores for angry faces were significantly above chance performance for the control group ( $p = .005$ ), but not for the DS group ( $p = .21$ ). For happy faces, percentage correct scores were above chance for control participants ( $p < .001$ ) and for DS patients ( $p = .005$ ). For neutral faces, percentage correct scores did not differ from chance in the DS group ( $p = .069$ ) or the controls ( $p = .02$ ), at the required alpha level.

Despite the findings that scores deviated from chance performance for some expressions, Table 30 indicates that the scores on the awareness test were far from 100% in all conditions, and that there was considerable range within groups, showing that some participants had very little awareness of the stimuli. In addition, subjective reports showed that 79% of the DS group and 72% of controls had no awareness of having seen any facial stimuli during the test at all. Those reporting awareness of having seen the stimuli often were not able to report exactly what they had seen (e.g. specific facial expressions).

**Table 30. Awareness check for subliminally presented stimuli**

|                                  | DS (n = 38)  | Control (n = 43) |
|----------------------------------|--------------|------------------|
| <b>Angry faces (% correct)</b>   |              |                  |
| Mean (SD)                        | 42.1 (24.2)  | 53.3 (24.7)*     |
| Median (IQR)                     | 40 (32.5)    | 60 (40)          |
| <b>Neutral faces (% correct)</b> |              |                  |
| Mean (SD)                        | 46.1 (23.3)  | 50.2 (25.9)      |
| Median (IQR)                     | 40 (30)      | 40 (40)          |
| <b>Happy faces (% correct)</b>   |              |                  |
| Mean (SD)                        | 52.9 (22.9)* | 60 (29.2)*       |
| Median (IQR)                     | 50 (30)      | 70 (50)          |

DS = dissociative seizures; SD = standard deviation; IQR = interquartile range

\* denotes variables significantly differing from chance performance ( $p < .01$ )

### 6.3.4. Emotional Stroop performance

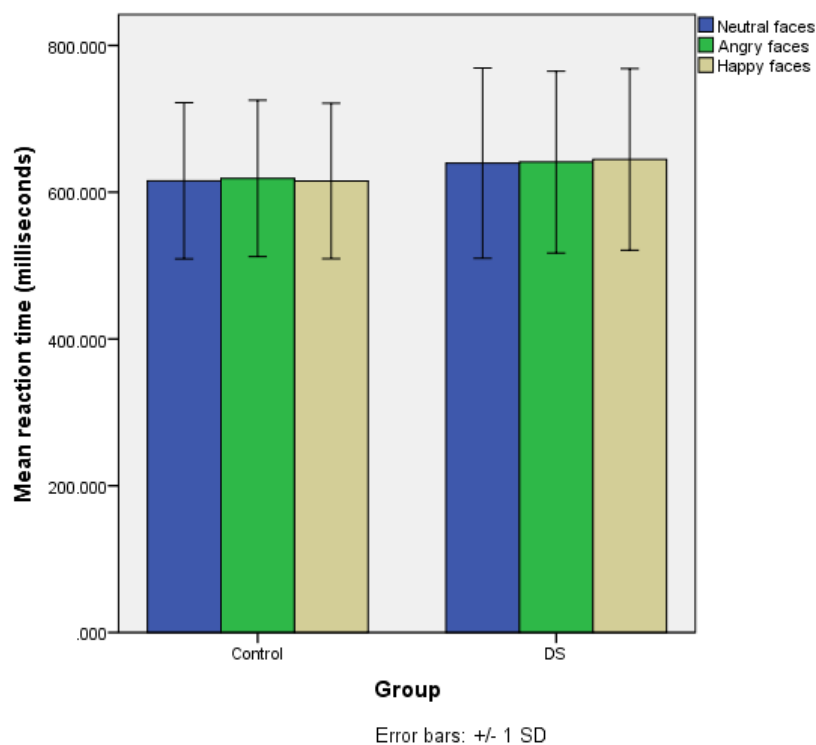
#### Error data

With YoE and HADS Anxiety and Depression scores included as covariates, the ANCOVA revealed no significant effect of group on error rates ( $F(1, 76) = 1.81, p = .183$ ); however, a significant effect of expression was evident ( $F(1.726, 152) = 8.104, p < .001$ ) with neutral expressions receiving most erroneous responses, and happy faces least. Nonetheless, there was still no significant interaction between group and expression ( $F(1.726, 152) = 1.11, p = .325$ ), indicating that the same pattern of errors was apparent across both groups.

#### Absolute RTs

Figure 9 displays the means and standard deviations for absolute RTs. For absolute RTs, after controlling for YoE and HADS scores (Anxiety, Depression), no significant main effect of expression ( $F(2, 152) = .391, p = .677$ ), group ( $F(1, 76) = .208, p = .650$ ), or interaction of group by expression ( $F(2, 152) = 2.61, p = .077$ ) was observed. None of the covariates were significant.

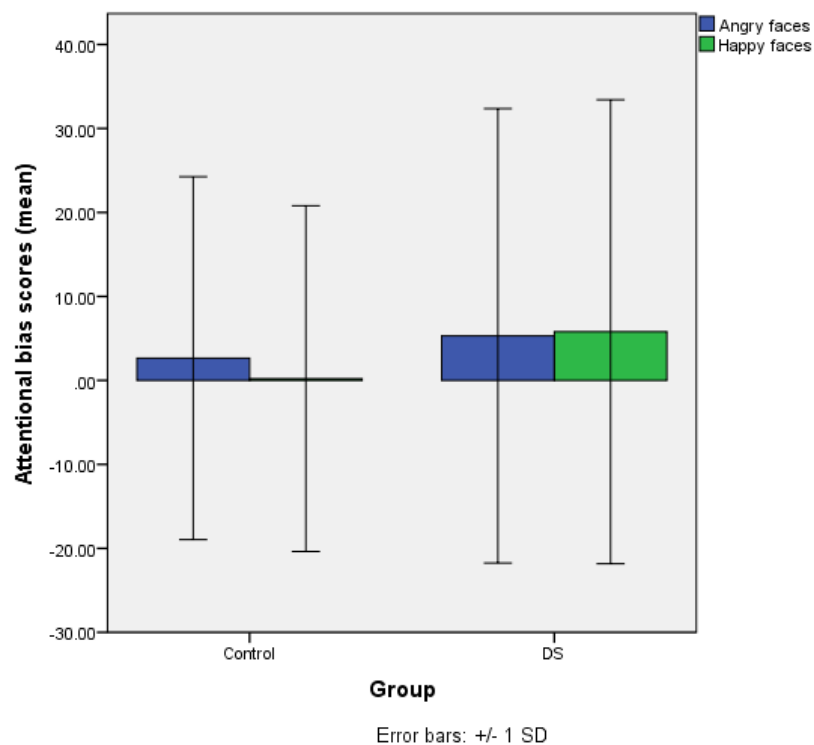
**Figure 9. Mean absolute reaction times by group and facial expression**



### AB scores

Figure 10 shows the descriptive statistics for AB scores. After covarying for YoE and HADS Anxiety and Depression scores, a significant between-groups effect was observed in attentional bias scores ( $F(1, 76) = 4.356, p = .04$ ), with the DS group showing significantly higher AB scores than control participants (see Figure 10). Within this analysis, there was no overall effect of expression ( $F(1, 76) = .830, p = .365$ ), and no expression by group interaction ( $F(1, 76) = .558, p = .458$ ). The only covariate that was significant was HADS Depression scores ( $F(1, 76) = 4.13, p = .046$ ), indicating that higher depression scores were associated with reduced AB scores. Furthermore, there were no significant interactions between expression type and any of the covariates.

**Figure 10. Mean attentional bias scores by group and expression**



In order to assess the possible influence of medication on AB scores, the ANCOVA was carried out twice more, with medication variables entered as additional between-group factors. When AED use was entered as an additional factor, neither the group

effect ( $F(1, 75) = 2.51, p = .117$ ) nor the effect of AED use ( $F(1, 75) = 2.53, p = .116$ ) were significant; however, HADS depression scores remained significant as a covariate ( $F(1, 75) = 4.98, p = .029$ ). When antidepressant use (yes/no) was entered as the additional factor, the group effect ( $F(1, 75) = 1.58, p = .213$ ) was not significant, and neither was the effect of antidepressant use ( $F(1, 75) = 3.58, p = .062$ ). However, HADS Depression was a significant covariate ( $F(1, 75) = 5.85, p = .018$ ).

### **6.3.5. Relationship of AB scores to participant characteristics**

With a stringent alpha level of  $p < .01$ , there were no significant simple correlations between any of the variables examined and AB scores for either type of facial expression. These analyses were carried out a second time using partial correlations controlling for YoE. In this second set of analyses, a significant positive relationship was observed between seizure frequency and AB scores for happy faces ( $r = .682, p < .001$ ). Again, no other variable was significantly associated with AB scores for either facial expression.

Due to the equivocal findings on the ANCOVAs for AEDs and antidepressants, point biserial correlations were conducted to examine whether the use of either AEDs or antidepressants was specifically related to AB scores for happy or angry faces separately. None of these relationships were significant at  $p < .01$ . However, whilst not meeting this more stringent alpha level, possible relationships were indicated between AB scores for happy faces and AED use ( $r_{pb} = .271, p = .014$ ) and antidepressant use ( $r_{pb} = .222, p = .046$ ).

In order to examine the possible influence of medication on AB scores for happy faces more closely, a multivariate linear regression was carried out in the DS group (no controls were using AEDs or antidepressants), with happy AB scores as the outcome variable. YoE, HADS Depression scores, AED and antidepressant use (yes, no), and seizure frequency were entered as the predictor variables in that order. Seizure frequency was included due to the finding that it was significantly associated with happy AB scores. In this analysis (see Table 31), the model only became significant in the last two steps, involving the addition of medication (AEDs,

antidepressants) and seizure frequency respectively. Both of these steps significantly added to the model.

**Table 31. Regression statistics for the predictive relationship of YoE, depression, medication (AED/antidepressants) and seizure frequency, on attentional bias scores for happy faces**

| n = 38             | <i>b</i> | Confidence Interval<br>(CI, 95%) | <i>SE B</i> | $\beta$ | p-value |
|--------------------|----------|----------------------------------|-------------|---------|---------|
| <b>Block 1</b>     |          |                                  |             |         | .599    |
| YoE                | .948     | -2.67, 4.57                      | 1.79        | .088    | .599    |
| <b>Block 2</b>     |          |                                  |             |         | .103    |
| YoE                | 1.15     | -2.39, 4.7                       | 1.75        | .107    | .513    |
| HADS Depression    | -1.77    | -3.93, .379                      | 1.06        | -.272   | .103    |
| <b>Block 3</b>     |          |                                  |             |         | .02     |
| YoE                | 2.15     | -1.18, 5.47                      | 1.63        | .199    | .198    |
| HADS Depression    | -2.49    | -4.52, -.453                     | .999        | -.381   | .018    |
| AED use            | 19.8     | .527, 39.1                       | 9.48        | .345    | .044    |
| Antidepressant use | 11.01    | -7.28, 29.3                      | 8.99        | .2      | .229    |
| <b>Block 4</b>     |          |                                  |             |         | .024    |
| YoE                | 1.71     | -1.43, 4.85                      | 1.54        | .159    | .276    |
| HADS Depression    | -2.09    | -4.03, -.154                     | .951        | -.32    | .035    |
| AED use            | 17.5     | -.755, 35.7                      | 8.94        | .304    | .06     |
| Antidepressant use | 5.69     | -12.1, 23.4                      | 8.72        | .103    | .519    |
| Seizure frequency  | .201     | .028, .374                       | .085        | .35     | .024    |

AED = anti-epileptic drugs; YoE = years of full-time education (or equiv)  
HADS = Hospital Anxiety & Depression Scale

The final model was highly significant ( $F(37) = 3.98$ ,  $p = .006$ ). With all variables entered, HADS Depression scores and seizure frequency remained significant, with the former being associated with lower AB scores and the latter being associated with higher AB scores. The influence of AED use was a non-significant trend in the final model. To summarise, when the possible influences of YoE, depression, and antidepressant use were taken into account, more frequent seizures and (to a lesser extent) AED use, were predictive of elevated AB scores for happy faces. However, the model accounted for only 38.4% of the variance in AB scores for happy faces.

## **6.4. Discussion**

### **6.4.1. Summary and interpretation**

This study attempted to replicate and extend the findings reported by Bakvis, Roelofs, et al. (2009); more specifically, the finding that patients with DS showed a specific preconscious attentional bias to threatening facial expressions (anger). Another aim was to replicate the finding that such an attentional bias is related to reports of sexual abuse in the DS group. Furthermore, the study was also designed to explore possible relationships between AB scores and other important psychosocial and seizure-related variables.

#### ***Awareness check***

Using a forced-choice procedure modelled on that used by Bakvis et al. (2009), the awareness check data indicated that there were no between-groups differences in awareness of the facial stimuli, suggesting that differences in awareness levels should not have influenced performance on the emotional Stroop test. However, both groups showed significantly better recognition of the stimuli than would be expected by chance, for one or more expressions. These findings suggested that some degree of awareness of the stimuli may have been present in a proportion of participants. This is in contrast to the findings of Bakvis, Roelofs, et al. (2009), who reported that their sample did not perform above chance level, in terms of awareness. It is, therefore, possible that presenting the faces for 17 milliseconds increased the degree of awareness of the stimuli in this study.

#### ***Emotional Stroop performance***

##### ***Absolute reaction times (RTs)***

The results indicated that there were no group, expression, or group/expression interaction effects on absolute mean RTs on the emotional Stroop task, after controlling for YoE and HADS scores. Therefore, relative to each other, the two groups showed comparable overall processing/response speed. This suggests that the differences in medication status between groups was not associated with an overall slowing of cognitive functioning (e.g. attention, processing speed) in the patient



sample. Moreover, it seems that none of the facial expressions (happy, angry, neutral) were associated with significant changes in overall processing/response speed across the entire sample.

#### *Attentional bias (AB) scores*

Having ascertained that there were no absolute differences in RTs during the task, calculation of the AB scores allowed quantification of the extent to which the emotional facial expression stimuli (i.e. happy, angry) affected performance, relative to the neutral faces. Thus, the relative effect of each emotional condition relative to the neutral condition was calculated for each individual and then averaged for each group.

After controlling for YoE, depression and anxiety, patients with DS showed a significantly greater attentional bias to the emotional faces, relative to the healthy control group. The lack of expression or interaction effects indicated that the pattern of performance was similar for each expression in both groups. Only depression scores were found to be a significant covariate in this analysis. This overall pattern of results suggested that, relative to control participants, and after the influences of YoE and depression were accounted for, patients with DS automatically allocated more attention to emotional faces compared to neutral faces. In light of the findings of Bakvis, Roelofs, et al. (2009), these findings support the hypothesis that patients with DS show some degree of hypervigilance for emotional facial expressions. This study, however, suggests that this hypervigilance might apply to both positive and negative facial expressions.

The finding that HADS Depression scores were a significant covariate in the analysis highlights the importance of examining the influence of these symptoms in studies of this nature. It is well-established that patients with DS often present with some degree of depression and/or anxiety (see Chapter 2, section 2.3.2). However, it is important to tease out what characterises the emotional processing style of patients with DS, independently of the possible influence of comorbid depressive or anxiety symptoms. Of particular note in this study was that higher depressive symptoms were associated with reduced attentional bias scores, suggesting that the presence of

depressive symptoms did not contribute to the elevated AB scores observed in the DS group. The exploratory regression analysis supported this suggestion (Table 31). It seems that in patients with DS, elevated depressive symptoms seem to be associated with some degree of cognitive avoidance of emotional facial expressions. This pattern contrasts with previous literature, in which an attentional bias towards negative stimuli has been indicated in people with depression (Peckham et al., 2010).

However, the analyses including AEDs and antidepressants as additional factors were somewhat difficult to interpret. The addition of each of these variables as additional between-group factors (in separate analyses) led to the group effect no longer remaining significant in each. Moreover, the effects of each drug type were not significant either. These findings suggested that there might have been medication effects on the AB scores, but it is possible that due to a lack of statistical power (only 13 patients were taking AEDs, for example), or other statistical considerations (e.g. inter-correlations between variables, unequal sample sizes for the medication variables), the effects of both group and medication were obscured. Therefore, these analyses did not allow a conclusive exclusion of possible medication effects, nor did they confirm that medication effects were entirely responsible for the group effects observed in the previous analyses. However, the fact that the absolute RTs were not significantly slower in the DS group suggested that medication in this group had not caused generally reduced cognitive processing speed.

With regard to the exploratory analyses with psychosocial and seizure-related variables, when YoE was controlled for, a significant relationship was observed between seizure frequency and AB scores for happy expressions. This finding suggests that those patients having the most seizures (which could be seen as a measure of disorder severity) preconsciously allocated more attention to positive facial expressions. It is possible that, as a consequence of experiencing higher levels of symptoms and the associated social/occupational limitations this might entail, patients experiencing more frequent seizures might preconsciously seek out positive experiences and interpersonal interactions. On the other hand, a pre-existing tendency to automatically attend to positive social cues or positive stimuli more generally might serve as a means of reducing, avoiding or coping with emotional

distress. This tendency to 'focus on the positive' in the environment, or to be hypervigilant to signs of social approval, could be a psychological mechanism that somehow contributed to the development of DS. Obviously, these interpretations are speculative and would require further examination in more focused studies.

Importantly, the correlational analyses further examined the possible influence of relevant medications. These analyses indicated the possible influence of AED and antidepressant use on AB scores for happy expressions. When examined in the regression analysis in the DS group, it was apparent that AED use was associated with elevated AB scores for happy faces. However, when seizure frequency was also added to the model, the influence of AEDs became non-significant (although a trend remained) and seizure frequency was found to exert an independent influence on happy AB scores, beyond the influence of AEDs. This supports the above proposition, that the experience of having frequent seizures is uniquely associated with hypervigilance for happy faces in the DS group. It should be noted, however, that whilst the final regression model was significant, the amount of variance explained was modest (approximately 38%); therefore, it is clear that other factors possibly contributed to the AB scores.

Combined, the exploratory medication analyses suggested that AED use may have had some effect on AB scores in the DS group. AEDs are known to have cognitive side effects (e.g. Aldenkamp, Krom, & Reijs, 2003; Drane & Meador, 2002) and some (e.g. carbamazepine, valproate, lamotrigine) influence mood and/or behaviour (Drane & Meador, 2002; Hixson & Kirsch, 2009; Reijs, Aldenkamp, & Krom, 2004). The use of AEDs as psychotropic agents in the treatment of various psychiatric disorders is becoming more common (Nadkarni & Devinsky, 2005). It is, therefore, possible that AED use contributed to the group differences in AB scores, either by alterations in cognitive processes (e.g. attentional dysfunction), or by influencing mood state. On the other hand, it is possible that the effects of AEDs might be explained by a third variable, such as seizure severity or chronicity of the disorder. Future studies might examine the influence of AED use on emotional processing in larger samples of patients with DS or ES.

#### **6.4.2. Strengths and limitations**

An important strength of this study was the inclusion of a larger sample of patients with DS than that included by Bakvis, Roelofs, et al. (2009). This provided a satisfactory level of statistical power for the detection of possible group effects on performance on the emotional Stroop test. Recruitment of this sample size depended on relative inclusiveness during the recruitment process (e.g. patients diagnosed on the basis of clinical consensus and currently taking medication were included). On the one hand, this inclusiveness provided greater representativeness of the population being studied. However, this also leaves open the possibility that some patients may have comorbid ES or a misdiagnosis (although this possibility also remains in those diagnosed with video-EEG, albeit to a lesser extent).

An additional strength of the current study was the careful matching of the patient group with the control group. As outlined in section 6.3, the two groups were comparable on many important variables. Whilst there was an almost significant between-group difference in YoE, this was explicitly stated and taken into account within the statistical procedures carried out. This level of control allowed an insight into the extent to which YoE affected performance on the experimental task, but also to examine the group differences once this possible confound had been statistically controlled for.

A related issue is that relevant standardised cognitive measures were included in the test battery of the current study; therefore, it was possible to conclude with some degree of confidence that the groups were matched on general intellectual functioning and processing/recognition of faces, in addition to comparable performance on a standard Stroop test. This allowed clearer interpretation of the findings on the experimental task, and ensured that they could not have been better explained by deficits in the measured cognitive domains. Given the difference in YoE between groups, and the fact that YoE is generally highly correlated with IQ scores, the inclusion of the WASI was very important, in order to ensure that a significant difference in intellectual functioning was not present. Furthermore, the exploratory evaluation of possible relationships between the psychosocial and seizure-related variables and AB scores, provided greater depth to the study. Moreover, because of

the inclusion of a measure of anxiety and depression (HADS), it was also possible to examine the influence of general emotional distress (i.e. depression, anxiety) on AB scores, and to control for its influence.

One key limitation of the current study was the possibility of medication effects; however, this will be discussed further in Chapter 10 as it also applies to the other experimental studies. Furthermore, whilst there were no between group differences in awareness of the experimental stimuli in this study, both groups performed better than chance for one or more facial expressions, on the forced-choice awareness procedure. This suggested that the 17 millisecond duration presentation time for the facial stimuli did not satisfactorily preclude conscious awareness of them. Whilst participants were not directly and explicitly required to attend to the facial expressions, it is possible that some residual awareness was present.

As mentioned, the exploratory correlational analyses yielded very few results that met the required significance level. Furthermore, the relationship between AB scores and sexual abuse reported by Bakvis et al. (2009) was not replicated in this study. It is possible that the adoption of a stringent alpha level ( $p < .01$ ) may have led to Type 2 errors in this regard; however, this was thought to be a necessary precaution to avoid the undesirable increase in risk of Type 1 errors caused by a large number of related tests. Future work may focus on fewer variables, in order to reduce the requirement for such a stringent alpha level. Further research in this area might also seek to include a larger sample size, so that a comparison of larger subgroups of medicated and unmedicated participants might be compared, whilst controlling for relevant confounds (anxiety, depression, cognitive functioning, education). This would allow greater statistical power to be retained when examining the influence of these possible confounds.

#### **6.4.3. Summary and conclusions**

The results of the current study are partially supportive of the findings reported by Bakvis, Roelofs, et al. (2009). After controlling for YoE, anxiety and depression, an overall group effect on AB scores was observed (higher scores in the DS group). However, this elevation was not specific to angry faces in the current study, as it was

in that of Bakvis, Roelofs, and colleagues (2009). Moreover, depression was found to covary with AB scores in this study, indicating that depressive symptoms in DS might influence preconscious emotional processing in a different way to individuals with depression only. It was apparent that attentional biases for happy faces in the DS group were influenced by the frequency of patients' attacks, and to a lesser extent, the use of AEDs. Together, the results suggest that hypervigilance for emotional faces is likely to be characteristic of patients with DS, but that it is of considerable importance to measure and examine the relative influence of possible confounding variables, including current psychopathology, medication use, and the severity of the disorder.

## **Chapter 7. Conscious facial expression processing in patients diagnosed with dissociative seizures: subjective and autonomic responding**

### **7.1. Introduction**

#### **7.1.1. Background**

In 'The Expression of the Emotions in Man and Animals' (1872), Charles Darwin discussed the nature of emotional expressions across species and proposed that they are genetically determined and a result of natural selection. Moreover, he argued that some emotional expressions are universal in human beings. In the twentieth century, Paul Ekman and his colleagues gathered empirical evidence for the universality of facial expressions, identifying several that were expressed and recognised consistently across literate and preliterate peoples (e.g. Ekman & Friesen, 1971). These expressions included anger, disgust, fear, happiness, surprise and sadness, and were later termed 'basic' emotions (Ekman, 1992). Subsequent research has generally supported the universality hypothesis (Elfenbein & Ambady, 2002; Matsumoto et al., 2008). The ability to recognise facial expressions of some emotions, therefore, seems to be a 'hard-wired' adaptation that likely served an important survival-related function in the evolutionary history of the species. Probably the most important function is that of social communication. Others' facial expressions convey important information about their intentions towards us, but also about other significant stimuli in the environment. Accurate and efficient interpretation of others' emotional facial expressions is particularly important for successful social interaction, and the ability to form and maintain relationships.

Experimental evidence has indicated that conscious or explicit processing of facial expressions can be affected in several psychological disorders that share similarities, or are commonly comorbid with, DS. For example, there is a large empirical literature on facial expression processing in individuals with affective disorders, and whilst there are mixed findings, a meta-analysis including 51 studies (Kohler et al., 2011) found evidence for moderate impairments in facial expression recognition in people diagnosed with major depression and bipolar disorder. A number of variables were reported to be associated with reduced performance, including more severe

subjective symptoms of depression, younger age at testing, and fewer years of education. These findings suggest the importance of measuring and controlling for variables such as symptom severity, age and educational history, in studies of this nature.

Individuals diagnosed with PTSD have also been reported to show reduced sensitivity to and accuracy in recognising fear and sadness (e.g. Poljac, Montagne, & de Haan, 2011), and to perceive higher levels of negativity in some basic facial expressions relative to traumatised controls without PTSD (Shin et al., 2005). A recent meta-analysis (Plana et al., 2014) summarised studies of emotion recognition in patients with PTSD and other anxiety disorders and concluded that, whilst deficits have been observed across the anxiety disorders, the effect size for emotion recognition deficits in PTSD is larger than in other anxiety disorders, such as generalised or social anxiety.

A wide range of studies indicates that emotion recognition may also be atypical in patients diagnosed with borderline personality disorder (BPD). Most consistently, reduced accuracy in recognition of emotional facial expressions has been reported in patients with BPD compared to controls (Bland et al., 2004; Levine, Marziali, & Hood, 1997; Merkl et al., 2010; Nicol, Pope, & Hall, 2014; Unoka et al., 2011), although there have been differences in the extent of the reported deficits (i.e. how many emotions are affected). Conversely, Wagner & Linehan (1999) and Lynch et al. (2006) reported increased rather than decreased sensitivity for detection of emotional facial expressions in patients with BPD relative to controls. Furthermore, Minzenburg, Poole, and Vinogradov (2006) only observed deficits in recognition of combined prosodic and facial emotional expressions, rather than in either of these independently. Despite the inconsistencies, Daros, Zakzanis, and Ruocco (2013) conducted a meta-analysis of studies assessing facial expression recognition in patients with BPD compared to healthy controls, and concluded that there was evidence for reduced recognition of specific negative emotions (i.e. anger, disgust) and neutral stimuli in the BPD group.

Some investigators have also explored facial affect recognition in patients with depersonalisation disorder (DPD). Montagne et al. (2007), for example, reported reduced recognition of angry facial expressions in patients with DPD relative to non-



clinical control participants, whereas Sierra and colleagues (2002) found that patients with DPD rated disgusted facial expressions as subjectively less intense relative to healthy and clinical (anxiety) control groups. In the latter study, the DPD group also exhibited reduced skin conductance responses to the disgusted facial expressions relative to the anxious group, and a similar trend relative to the healthy control group. It was interpreted that DPD provides a mechanism by which psychophysiological anxiety responses are inhibited or 'blunted' (p.229).

There have, however, been few studies of explicit facial expression processing in somatoform/conversion disorders. Pedrosa and colleagues (2009) observed reduced accuracy of recognition of facial expressions in individuals with somatoform diagnoses, relative to healthy control participants. This deficit was no longer significant when alexithymia scores were examined as a covariate, suggesting that emotion recognition difficulties were related to alexithymic tendencies in that group. Nonetheless, Pollatos and colleagues (2011) also examined facial expression recognition in patients with mixed somatoform diagnoses relative to healthy controls, reporting reduced recognition of sad and neutral faces, in addition to lower parasympathetic activity during the task. Moreover, lower ratings of emotional arousal in the emotional faces were also noted, relative to the control group.

Differences in findings within populations may well be attributable to variations in methodology between studies. For example, some investigators have included non-clinical traumatised individuals as control groups (Barnett Veague & Hooley, 2014; Wagner & Linehan, 1999), whereas others have included controls with unknown trauma history. Moreover, some studies include patients with comorbid psychiatric disorders, whereas others have excluded individuals with diagnoses other than that of particular interest in the study (e.g. Minzenburg et al., 2006). Another factor that can affect results in this area is the wide variety of different tasks used to examine facial expression recognition. The most common task involves a forced-choice procedure whereby participants select an emotional label from a limited selection of alternatives. However, Wagner & Linehan (1999), for example, used a test that allowed participants to freely choose the emotion label they ascribed to the faces. Moreover, Dyck et al. (2008) found that patients with BPD showed deficits on a time-constrained

task, but not on a task that did not include a time limit. Domes et al. (2008) also included a test which involved blends of emotions expressed in each face, with the ratio varying in a systematic manner. It was found that when the ratios were highly ambiguous (50:50), patients with BPD were more likely to perceive anger than controls, indicating a potential bias toward perceiving threat in uncertain social cues.

Studies also vary in the extent to which groups are matched on possible confounding variables, such as gender, age, ethnicity and educational background. For example, Levine et al. (1997) did not match groups for sex or marital status, so these factors may have influenced the results in this study. The gender of the models used in the facial stimuli may also influence the results in some clinical groups (Barnett Veague & Hooley; 2014). Furthermore, whilst there are exceptions (e.g. Montagne et al., 2007), many studies failed to include a measure of general face processing abilities. Therefore, in several studies, it is not possible to conclude that the observed emotion recognition deficits were not due to difficulties in perceptual processing of the faces.

### **7.1.2. Rationale, aims and hypotheses**

There are currently no published experimental studies of conscious (explicit) facial expression processing in patients with DS. As outlined in Chapter 3, there are experimental studies in which facial expressions have been included as distractor stimuli, and the findings suggest that automatic or preconscious processing of these stimuli may be altered. However, in none of these studies have patients been explicitly requested to attend to the emotional expressions, or to evaluate this feature consciously. Only one published report described an evaluation of explicit emotion recognition in patients with DS, which was incorporated within a standardised neuropsychological test battery (Prigatano & Kirlin, 2009). The group of patients with DS showed reduced overall performance on this subscale relative to patients with ES. However, because the subscale also included items on prosodic emotional expression, responses to a humorous stimulus and general affective control (assessed on the basis of the appropriateness of behaviour), the results were not clearly due to facial affect recognition deficits.

Therefore, the current experiment was designed to assess participants' subjective, conscious responses to facial expressions of emotion, when these were presented at durations ensuring conscious perception of the stimuli, and when the task explicitly required participants to evaluate the emotional expressions presented. Furthermore, the study sought to examine autonomic responding to the facial expression stimuli, by including a skin conductance measure. The term 'skin conductance response' (SCR) refers to a "...phasic, usually elicited, increase in skin conductance" (Lykken & Venables, 1971, p. 657), and this typically reflects increased activation of the sympathetic nervous system. The SCR is influenced by psychologically-mediated sweat production in the eccrine sweat glands (Dawson, Schell, & Filion, 2000), usually influenced by stimulus novelty, unexpectedness, intensity, and significance. As such, the SCR can be seen as part of an 'orienting response' (Dawson, et al., 2000). Whilst it is not possible to conclusively interpret the psychological significance of a positive SCR, higher levels of experimental control can reduce the uncertainty. In the present study, positive SCRs were interpreted as a sympathetic arousal response to the facial stimuli presented. This measure was included to establish whether similar or different patterns of responding would occur in the subjective and physiological domains. These processes were compared in patients with DS and healthy controls.

Another aim of the study was to carry out exploratory analyses examining the extent to which any differences in subjective or physiological responses to facial expressions presented in the current task were related to the preconscious attentional bias scores reported in Chapter 6. Moreover, a final objective was to assess whether relevant psychosocial and seizure-related variables in the DS group were associated with any observed differences in subjective or physiological responses to the stimuli.

On the basis of the literature reviewed above, the following hypotheses were tested:

- I. Patients with DS were expected to show reduced accuracy in recognition of emotional facial expressions, relative to the healthy control group. It was expected that this deficit would be specific to negative facial expressions.

2. Patients with DS were predicted to perceive increased intensity of emotion in facial expressions, relative to healthy control participants. Once again, this effect was expected for negative facial expressions only.

3. Differences in physiological responses (SCRs) to facial expressions of emotion were hypothesised to be observed in patients with DS, relative to healthy control participants. On the basis of the model proposed in Chapter 3 and previous findings, it was expected that patients would show higher levels of autonomic arousal compared to healthy controls, particularly for negative facial expressions.

In order to test these hypotheses, an experimental task was devised which involved the presentation of several types of facial expression, for a time period that allowed participants to intentionally and consciously process their emotional content (i.e. six seconds per stimulus). Dependent variables were participants' accuracy in categorising the expressions with emotion descriptors, subjective ratings of the intensity of the facial expressions, and SCRs. The responses of the patients with DS were compared to those of a healthy control group.

## **7.2. Methodology**

During the pilot study (see Chapter 4, section 4.1), it became clear that the facial expression processing task was too long (taking around 45 minutes). This was due to the number of levels of the within-subjects independent variable (facial expression type: happiness, anger, fear, disgust, neutral, sadness, surprise), plus the requirement of a relatively long ITI due to the psychophysiological data collection (see below). Therefore, the number of within-subjects conditions were reduced and the ITI was shortened. The final design is presented in Section 7.2.3.

### **7.2.1. Participants**

Details of recruitment procedures and eligibility criteria for this experiment were described in Chapter 4 (sections 4.2.1. and 4.2.2). Forty-three healthy control participants and 40 patients diagnosed with DS completed the experiment.

### **7.2.2. Materials**

#### ***Experimental materials***

The stimuli used in this task were taken from a standardised set of grey-scale images of actors pulling a variety of facial expressions (Pictures of Facial Affect; Ekman & Friesen, 1976). Three of the 14 actors included in the set had not posed a fearful facial expression; therefore, images of these actors were not used in the task. Of the remaining images, normative ratings were examined and the images of the six (three male, three female) actors with the highest overall recognition rates were selected as the experimental stimuli.

Initially, all seven facial expressions were included (i.e. happiness, sadness, fear, anger, disgust, surprise, neutral). The faces were digitally cropped for standardisation and to focus attention specifically on the facial expression. The cropped faces were positioned centrally against a black background, to maximise contrast. Examples are shown in Appendix 14. On completion of the pilot study, the number of facial expression types was reduced from seven to five, by removing the sadness and surprise conditions. The removal of 12 stimuli from the task allowed a reduction of approximately 20 minutes for its completion.

#### ***Skin conductance recording apparatus***

SCR data were gathered using a Powerlab data acquisition system (ADInstruments) which was connected to the laptop, and attached to a 'Galvanic Skin Response' Amplifier (ADInstruments). Stainless steel field electrodes were used to obtain SCR recordings. The use of dry electrodes (i.e. no electrolyte gels) was recommended with the particular amplifier used, because of the application of a low constant voltage ( $22\text{mV}_{\text{rms}}@75\text{Hz}$ ; ADInstruments, 2008). SCRs were recorded online with LabChart software (v6.0, ADInstruments). The experimental software (E-Prime) and LabChart software were programmed to interact, in order that markers could be placed onto the SCR traces for each individual stimulus. A parallel port card drive (PCMCIA, Quatech.com) and custom-made cable (MLACX, ADInstruments) were used to connect the Powerlab and laptop to allow this interaction.

### ***Emotion label comprehension check***

Prior to commencing the experimental task, participants' comprehension of the emotional descriptors was checked. Participants were asked to describe the experience of each emotional state (e.g. happy, fearful, angry, neutral, disgusted). If participants found this difficult, they were asked to give an example of a situation in which they might experience that emotional state. The responses were recorded by the investigator.

### ***Neuropsychological measures***

*Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999)*

As described in Chapter 6 (section 6.2.2.), the two subscale form of this test was administered to assess current intellectual functioning.

*Benton Facial Recognition Test (BFRT; short-form; Benton et al., 1983)*

Chapter 6 (section 6.2.2) provides a description of this test. It was included to assess general perceptual processing of facial stimuli.

*Wechsler Memory Scale – Third Edition (WMS-III; Wechsler, 1997)*

The Faces I subtest of the WMS-III was administered to measure short-term memory for facial stimuli (recognition). The facial expression processing task in the current study required participants to retain facial stimuli in short-term memory, in order to provide subjective responses during the ITI. Therefore, measuring (or controlling for) short-term memory for such stimuli is important, to ensure that group differences on this variable did not influence performance on the facial expression processing task. The raw scores on the Faces I subtest can be transformed into scaled scores on the basis of age, which can then be compared to normative comparison groups.

### **7.2.3. Design**

The experiment had a mixed factorial design, with one between-groups factor (diagnostic status: DS, control) and one within-groups factor (facial expression: anger,

happiness, fear, disgust, neutral). The dependent variables were subjective ratings (selection of emotion descriptor from multiple-choices, ratings of emotion intensity) and SCRs. Several measures of SCR were examined, including amplitude and response frequency. These measures are outlined further below (section 7.2.5). There were six examples of each facial expression yielding a total of 30 stimuli (trials). A list of the stimuli included in the final design can be found in Appendix 14. The stimuli were presented in a novel pseudo-random order for each participant. No more than two stimuli with the same expression were presented consecutively.

#### **7.2.4. Procedure**

Participants were seated approximately half a metre from the laptop computer, placed on a desk. At the start of the session, the SCR electrodes were attached and participants were asked to relax and sit quietly during a five-minute habituation period. Following the five minute rest period, participants' comfort and well-being was checked and any necessary amendments were made (e.g. loosening sensors slightly, altering chair). When participants were ready, the experimental task commenced. Standardised instructions (Appendix 15) were presented on the computer screen, and read aloud by the experimenter. The instructions were presented in white font (size 16-18; Tahoma style) on a black screen. Any questions were answered at this time.

Once participants were satisfied that they understood the task, they completed three practice trials, including pictures of the same actor posing happiness, disgust and neutrality (images of this actor were not included in the experimental trials). After participants had completed the practice trials and asked any remaining questions, they commenced the experimental trials. The experimental trials and practice trials were identical. Each trial started with a 15-second presentation of a central white fixation cross, presented against a black background. This duration of ITI was selected in order to ensure that SCRs elicited by each stimulus were clearly distinguishable (Boucsein et al., 2012).

Following offset of the fixation cross, a face showing one of the target emotions was presented for six seconds. This presentation time was selected because SCRs

typically reach their peak between one and four seconds after stimulus onset (Boucsein et al., 2012; Dawson et al., 2000). Immediately after stimulus offset, participants were presented with a screen asking them to select which of the five emotions was expressed in the face, with numbers next to each descriptor indicating how participants could make their choices (see Appendix I6 for a screenshot). The order of the multiple options was randomised on each trial. After the participant chose a descriptor, the second rating screen appeared (Appendix I6). This required that participants judge how intense the emotion had been, on a scale from 0-7. After a response was registered, the fixation cross appeared again, starting the ITI. At the end of the task, a thank-you screen appeared and any questions were answered.

#### **7.2.5. SCR data acquisition, extraction and reduction**

All participants were requested to avoid smoking and consuming caffeine in the hour preceding the start of the research session. The SCR electrodes were placed on the distal phalanges of the index and middle fingers of the non-dominant hand. The distal phalanges were selected as SCR amplitudes may be greater at these sites (Scherbo et al., 1992). In accordance with Boucsein et al. (2012), no specific pre-treatment of the hands was carried out.

A constant voltage ( $22\text{mV}_{\text{rms}}$ ) was applied. SCR was sampled at 100 Hz. A 1Hz (second-order, low-pass) filter was applied to reduce noise in the signal. The SCR signal was calibrated for each participant prior to the experimental task, in order to detect a range from 0-50 microSiemens ( $\mu\text{S}$ ). Prior to data extraction, the SCR traces for all trials were visually inspected for obvious noise and/or artefact. In the context of biosignal processing, Gratton (2000, p. 912) defines noise as “...any phenomena observed in the data other than the signal(s) of interest to the investigator.” Any trials that included clear noise/artefact were excluded from the analysis. For SCR data, these would include non-specific skin conductance changes during any baseline period or extreme increases in skin conductance suggestive of movement, respiratory, or other physiological artefact.

Baseline values for each stimulus were calculated from the mean values during the one second immediately prior to stimulus onset, following M.M. Bradley et al. (2001),



for example. The maximum value (microSiemens;  $\mu\text{S}$ ) occurring between one and four seconds after stimulus onset was taken as the peak SCR amplitude for each trial (Dawson et al., 2000). Any trials in which there was a decrease in amplitude or a value of  $0\mu\text{S}$  were assigned a value of  $0\mu\text{S}$ .

The amplitude of SCRs for a given stimulus type can be taken as the mean of the positive responses to that stimulus type, excluding values of zero (Dawson et al., 2000). Responses of  $.01\mu\text{S}$  or greater were coded as a positive SCR and these values were included in an analysis of SCR amplitude. A minimum of between  $.01$  and  $.04\mu\text{S}$  is considered a positive SCR (Boucsein et al., 2012; Dawson et al., 2000). The value of  $.01\mu\text{S}$  was adopted here in order to reduce the likelihood of excluding genuine SCRs from the results. Positive amplitude values were averaged for each facial expression type, by participant. These values were then used to calculate average SCR amplitudes for each facial expression type, by group.

Furthermore, the percentage of valid trials (i.e. trials not including noise/artefact) in which a positive SCR occurred (i.e. values over  $.01\mu\text{S}$ ) was calculated for each participant by facial expression type, and averaged by group. This provided a measure of the frequency with which positive SCRs were observed for each facial expression, by group. Finally, for each participant, dichotomous coding classified whether they had shown one or more positive SCRs for each facial expression (i.e. response, no response). This yielded a more absolute measure of responsivity, by facial expression type and group.

#### **7.2.6. Statistical analysis**

##### ***Subjective measures***

Mean accuracy scores (0-6) were entered as the dependent variable in a mixed factorial ANCOVA, with group (DS, control) as the between-subjects variable and facial expression (happiness, anger, disgust, fear, neutral) as the within-subjects variable. The covariates were YoE, and HADS Anxiety and Depression scores. The mean intensity scores were entered as the dependent variable in a separate but similarly constructed analysis.

### ***Skin conductance responses***

SCR amplitude and magnitude data were analysed separately with mixed factorial ANCOVAs as described for the subjective ratings. The percentage of trials in which positive responses were observed was also entered as the dependent variable in a similarly constructed analysis. Chi-square tests were used to assess the relative proportion of each group showing at least one positive SCR for each facial expression.

### ***Exploratory analyses***

Exploratory correlational analyses were carried out to assess potential relationships between background psychosocial factors and any significant dependent variables in the facial expression processing task. The psychosocial variables included were those which had been found to differ between groups (see Chapter 5, section 5.3) as follows: the MDI (Disengagement, Depersonalisation, Derealisation, Emotional Constriction, Memory Disturbance, Identity Dissociation subscales), SDQ-20, IASC (Abandonment Concerns, Identity Impairment, Affect Dysregulation, Tension Reduction Activities subscales), HADS (Anxiety, Depression subscales), TEC (total scores, sexual abuse, physical abuse, mean impact scores), and PDS (Total, Re-experiencing, Avoidance, and Arousal scores).

In addition, seizure-related variables were included in the correlational analyses, to examine whether facial expression processing alterations might be associated with specific aspects of patients' seizures. These variables included seizure frequency, duration of the disorder (DS), and ictal symptoms (total, cognitive, mental state, chest abdomen, autonomic arousal, general). Furthermore, the use of AEDs and antidepressants were also entered into correlations with the dependent variables, to assess possible influences on performance. Pearson's or Spearman's correlations were used, depending on the distribution of scores. Two-tailed tests were used and alpha levels were set to  $p < .01$  to reduce the likelihood of type I errors caused by multiple testing. Any significant simple correlations were carried out a second time as partial correlations controlling for YoE, to check if the relationships remained significant after educational history had been taken into consideration.

## **7.3. Results**

### **7.3.1. Participant characteristics**

Demographic details and clinical characteristics are as described in Chapter 5 (Tables 2-5). As previously described, there was a between-groups difference in years of education (YoE); therefore, YoE was entered as a covariate in all analyses described in the present chapter. The groups did not significantly differ on any other demographic variable, although there was a trend for more DS patients to be in long-term relationships compared to controls. A greater proportion of the DS group were also taking medication and had medical diagnoses, relative to the control group.

### **7.3.2. Neuropsychological measures**

A summary of the statistics for the neuropsychological measures can be found in Table 32. There were no significant differences in full-scale IQ scores between-groups. The mean score for both groups fell within the average range, based on the normative data for this test. There were also no overall group effects on Vocabulary or Matrix Reasoning subtest scores. Furthermore, there were no between-groups differences on the Faces I subtest of the WMS-III, with the mean scores for each group indicating average performance on this measure.

However, there was a borderline significant between-group difference on the BFRT, with the DS group performing worse than the control group. Whilst the median and mean scores for each group were in the average range, closer examination of the data indicated that a small number of participants in each group scored within the borderline/impaired range on this measure (control = 1; DS = 4). Once these participants were excluded from the analysis, no significant differences were observed in BFRT scores, or any of the other neuropsychological measures.

In order to ensure that any potential differences on the experimental task could not be accounted for by poor perceptual processing of the facial stimuli, the main analyses were run twice, once including these participants, and once when these participants were excluded. If the results differed on exclusion of these participants, this is reported in the relevant section below.

**Table 32. Neuropsychological findings (facial expression processing task)**

|                                   | <b>DS</b>    | <b>Control</b> | <b>Test statistic (df)</b> | <b>p-value</b> |
|-----------------------------------|--------------|----------------|----------------------------|----------------|
| <b>WASI</b>                       | n = 40       | n = 43         |                            |                |
| FSIQ (Mean, SD)                   | 103.6 (14.5) | 108.1 (13.1)   | t (81) = 1.5               | p = .137       |
| Vocabulary (Mean, SD)             | 51.6 (11.1)  | 55.2 (9.8)     | t (81) = 1.6               | p = .118       |
| Matrix reasoning                  |              |                |                            |                |
| Mean (SD)                         | 52.1 (8.9)   | 53.7 (9.5)     | U (83) = 746               | p = .298       |
| Median (IQR)                      | 54 (10)      | 56 (15)        |                            |                |
| <b>BFRT (all participants)</b>    | n = 39       | n = 43         |                            |                |
| Mean (SD)                         | 46.9 (4.4)   | 48.9 (3.5)     | U (82) = 635               | p = .056       |
| Median (IQR)                      | 47 (7)       | 49 (5)         |                            |                |
| <b>BFRT (minus scores &lt;40)</b> | n = 35       | n = 42         |                            |                |
| Mean (SD)                         | 47.9 (3.6)   | 49.1 (3.2)     | U (77) = 598.5             | p = .158       |
| Median (IQR)                      | 49 (7)       | 49 (5)         |                            |                |
| <b>WMS-III</b>                    | n = 39       | n = 43         |                            |                |
| Faces scaled scores (Mean, SD)    | 10.9 (3.2)   | 11.1 (2.9)     | t (80) = .285              | p = .776       |

DS = dissociative seizures; SD = standard deviation; df = degrees of freedom; IQR = interquartile range; WASI = Wechsler Abbreviated Scale of Intelligence; FSIQ = Full-scale Intelligence Quotient; BFRT = Benton Facial Recognition Test; WMS-III = Wechsler Memory Scale – Third Edition

### 7.3.3. Emotion label comprehension check

All participants were able to provide definitions of the emotion labels, or gave relevant examples of situations that might trigger or be associated with each emotion. This suggested that all participants had acceptable and comparable levels of understanding of the emotion descriptors.

### 7.3.4. Subjective ratings

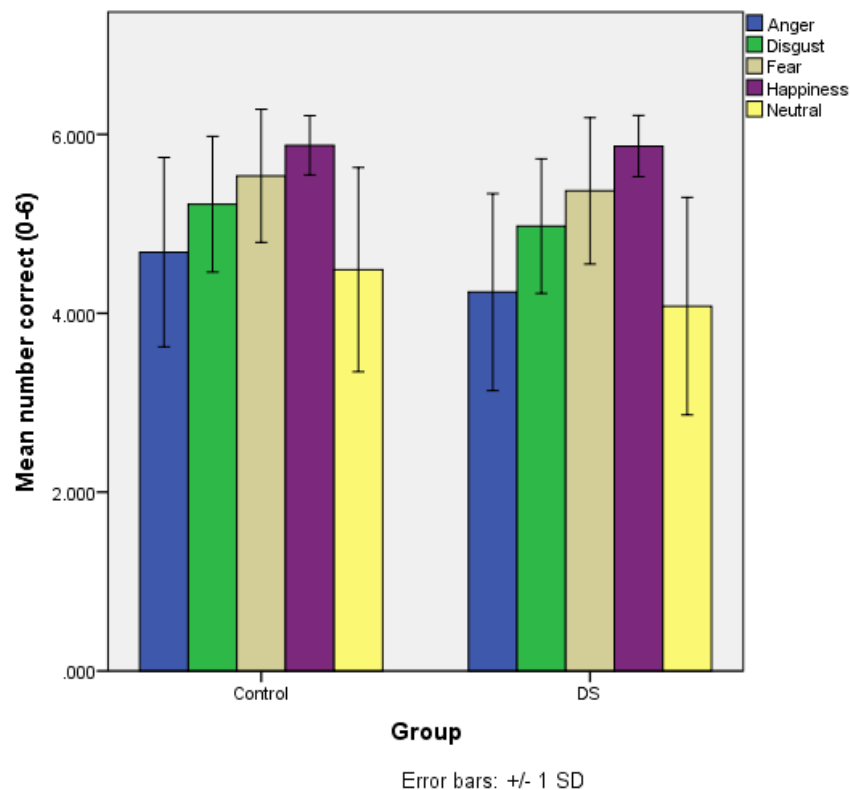
#### **Recognition accuracy**

Two participants in each group had outlying scores on one or more condition and their scores influenced the results; therefore, these participants were excluded from the analyses. Figure 11 displays the descriptive statistics for accuracy scores. The mixed factorial ANCOVA revealed that after covarying YoE and HADS scores (Anxiety and Depression), there were significant main effects of expression ( $F(3.38,$

250.4) = 4.19,  $p = .005$ ) and group ( $F(1, 74) = 8.56, p = .005$ ) on accuracy. Closer inspection of the estimated marginal means indicated inferior performance in the DS group (mean = 4.85, standard error = .079, 95% CI = 4.69, 5.01), compared to the control group (mean = 5.21, standard error = .075, 95% CI = 5.06, 5.36). The effects also remained significant when participants scoring in the borderline or impaired range on the BFRT were removed from the analysis (expression at  $p < .05$ , group at  $p < .01$ ).

Across all participants, neutral faces were recognised least accurately (marginal mean = 4.82, standard error = .134, 95% CI = 4.02, 4.55) and happy faces were recognised most accurately (mean = 5.87, standard error = .037, 95% CI = 5.8, 5.95). There was no interaction between expression and group ( $F(3.38, 250.4) = .695, p = .573$ ). Moreover, YoE ( $F(1, 74) = 2.17, p = .145$ ), HADS Anxiety ( $F(1, 74) = 1.13, p = .292$ ) and HADS Depression scores ( $F(1, 74) = .245, p = .622$ ) were not significant covariates in the analysis.

**Figure 11. Accuracy of facial expression recognition by group and facial expression**



In order to examine the effects of medication on accuracy scores, the ANCOVA was rerun twice, with AED use and antidepressant use entered as additional between-group factors respectively. The group effect remained significant ( $F(1, 73) = 6.1, p = .016$ ) after entering AED use in the analysis; however, the effect of AED use was not significant ( $F(1, 73) = .865, p = .356$ ). When antidepressant use was added to the ANCOVA, the group effect was significant ( $F(1, 73) = 6.94, p = .01$ ), whereas the effect of antidepressants was not ( $F(1, 73) = .057, p = .811$ ).

### ***Perceived intensity***

Descriptive statistics for the intensity ratings can be found in Figure 12. For intensity ratings, there was a significant effect of expression ( $F(1.74, 134.2) = 7.22, p = .002$ ), but no main effect of group ( $F(1, 77) = .131, p = .719$ ), and no group by expression interaction ( $F(1.74, 134.2) = 2.34, p = .107$ ). Neither HADS Anxiety ( $F(1, 77) = .03, p = .862$ ) nor Depression scores ( $F(1, 77) = .091, p = .764$ ) were significant covariates; however, YoE was a significant covariate ( $F(1, 77) = .463, p = .034$ ). Regarding the expression effect, the perceived intensity in neutral expressions was lowest and the perceived intensity of fear was rated highest.

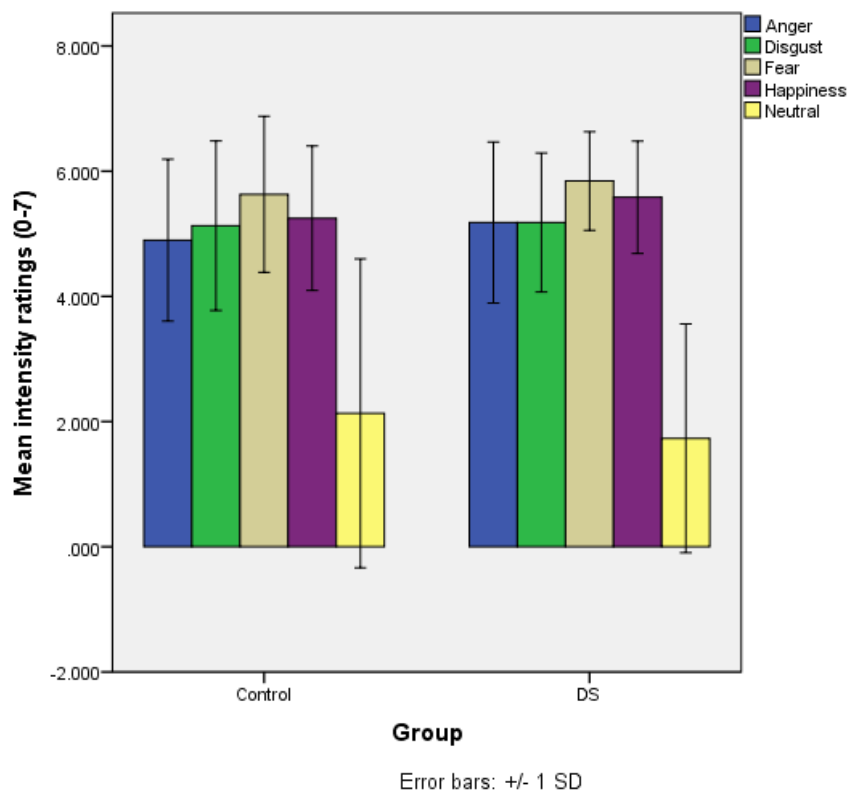
### **7.3.5. Skin conductance responses**

SCR data for four control participants were not usable due to technical failures or participants' behaviour (e.g. excessive movement artefact). Table 33 displays the descriptive statistics for all SCR variables described below.

### ***Total trials included***

Due to data loss associated with the exclusion of trials in which noise/artefact were observed, it was important to establish whether there were any systematic differences in the number of trials retained for analysis, by group or expression. A mixed factorial ANOVA showed that there were no group ( $F(1, 77) = .482, p = .49$ ) or facial expression ( $F(3.12, 240.5) = .862, p = .465$ ) effects on the number of trials retained in the analysis.

**Figure 12. Perceived emotional intensity ratings by group and facial expression**



### **Positive SCRs**

With YoE and HADS scores (Anxiety and Depression) as covariates, there were no significant main effects of group ( $F(1, 74) = .062, p = .804$ ) or facial expression ( $F(4, 296) = 1.52, p = .196$ ) on the percentage of (retained) trials on which positive SCRs were observed. The interaction between facial expression and group was also non-significant ( $F(4, 296) = .406, p = .805$ ). YoE ( $F(1, 74) = 1.03, p = .314$ ), HADS Depression ( $F(1, 74) = 1.52, p = .222$ ), and HADS Anxiety scores ( $F(1, 74) = .005, p = .943$ ) were not significant covariates.

Chi-square tests showed that there were no significant differences in the percentage of participants in each group showing at least one positive SCR for anger ( $\chi^2(1, 79) = 1.57, p = .210$ ), disgust ( $\chi^2(1, 79) = .027, p = .869$ ), fear ( $\chi^2(1, 79) = 1.13, p = .288$ ), happiness ( $\chi^2(1, 79) = .111, p = .739$ ) and neutral ( $\chi^2(1, 79) = .412, p = .521$ ). Moreover, there were no group differences in the proportion of autonomic 'responders' in each group, defined as those participants showing at least one positive SCR in every condition ( $\chi^2(1, 79) = .01, p = .922$ ).

**Table 33. Skin conductance response (SCR) descriptive statistics (facial expression processing task)**

|                                                                                    | <b>n</b>                | <b>DS</b>                                                                                                                                                                                                                                          | <b>Control</b>                                                                                                                                                                                                                                     |
|------------------------------------------------------------------------------------|-------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Total number of trials retained for analysis (0-6) (Median, IQR) (Mean, SD)</b> | DS = 40<br>Control = 39 | Anger: Mdn = 6 (1); Mean = 5.53 (.716)<br>Disgust: Mdn = 6 (1); Mean = 5.5 (.817)<br>Fear: Mdn = 6 (1); Mean = 5.63 (.667)<br>Happiness: Mdn = 6 (1); Mean = 5.6 (.744)<br>Neutral: Mdn = 6 (1); Mean = 5.63 (.628)                                | Anger: Mdn = 6 (1); Mean = 5.66 (.621)<br>Disgust: Mdn = 6 (1); Mean = 5.51 (1.1)<br>Fear: Mdn = 6 (1); Mean = 5.67 (.662)<br>Happiness: Mdn = 6 (1); Mean = 5.6 (.628)<br>Neutral: Mdn = 6 (0); Mean = 5.74 (.595)                                |
| <b>Percentage of trials with positive SCRs (0-100%) (Median, IQR) (Mean, SD)</b>   | DS = 40<br>Control = 39 | Anger: Mdn = 20 (50); Mean = 28.6 (25.7)<br>Disgust: Mdn = 22.5 (33.3); Mean = 23.7 (21.3)<br>Fear: Mdn = 16.7 (29.2); Mean = 23.3 (20.6)<br>Happiness: Mdn = 18.3 (16.7); Mean = 25.3 (19.5)<br>Neutral: Mdn = 20 (23.3); Mean = 28.7 (20.6)      | Anger: Mdn = 33.3 (33.3); Mean = 31.8 (21.4)<br>Disgust: Mdn = 16.7 (33.3); Mean = 23.03 (21.8)<br>Fear: Mdn = 20 (16.7); Mean = 27.9 (20.1)<br>Happiness: Mdn = 33.3 (33.3); Mean = 29.9 (24.2)<br>Neutral: Mdn = 33.3 (33.3); Mean = 32.5 (24.3) |
| <b>Percentage of participants displaying &gt;1 positive SCR n (% of group)</b>     | DS = 40<br>Control = 39 | Anger: 28 (70%)<br>Disgust: 27 (67.5%)<br>Fear: 30 (75%)<br>Happiness: 32 (80%)<br>Neutral: 34 (85%)                                                                                                                                               | Anger: 32 (82.1%)<br>Disgust: 27 (69.2%)<br>Fear: 33 (84.6%)<br>Happiness: 30 (76.9%)<br>Neutral: 31 (79.5%)                                                                                                                                       |
| <b>SCR amplitude in microSiemens (Median, IQR) (Mean, SD)</b>                      | DS = 16<br>Control = 16 | Anger: Mdn = .169 (.291); Mean = .232 (.238)<br>Disgust: Mdn = .222 (.465); Mean = .363 (.376)<br>Fear: Mdn = .17 (.281); Mean = .323 (.423)<br>Happiness: Mdn = .097 (.242); Mean = .164 (.147)<br>Neutral: Mdn = .326 (.599); Mean = .395 (.359) | Anger: Mdn = .29 (.824); Mean = .607 (.822)<br>Disgust: Mdn = .303 (.526); Mean = .514 (.767)<br>Fear: Mdn = .553 (.821); Mean = .71 (.859)<br>Happiness: Mdn = .564 (1.05); Mean = 1.05 (1.4)<br>Neutral: Mdn = .349 (.632); Mean = .528 (.517)   |

SD = standard deviation; IQR = interquartile range; Mdn = median



### **Amplitude**

Typically, log or square root transformations are used to normalise SCR values (Boucsein et al., 2012; Dawson et al., 2000). In this instance, neither of these transformations successfully normalised the SCR amplitude values for all conditions. Moreover, removal of two cases with outlying values on one or more condition (one from each group) also did not normalise the distribution of the data. Nonetheless, the planned factorial ANCOVA was conducted (see Chapter 4, section 4.5.3).

The results of the analyses were considerably different when the two outliers were excluded; therefore, the results stated below refer to those obtained with the outliers excluded. Due to the removal of the outlying participants and those with a score of zero on any one condition, this analysis was carried out with a reduced sample size (DS  $n = 16$ ; control  $n = 16$ ). These participants can be called ‘responders’.

The ANCOVA showed that there was no main effect of facial expression ( $F(3.2, 86.4) = .417, p = .754$ ) and no interaction between expression and group ( $F(3.2, 86.4) = 1.65, p = .181$ ). YoE ( $F(1, 27) = .031, p = .861$ ), HADS Anxiety ( $F(1, 27) = .333, p = .569$ ) and Depression ( $F(1, 27) = .022, p = .883$ ) were not significant covariates. However, there was a borderline significant main effect of group ( $F(1, 27) = 4.04, p = .055$ ), with the DS group showing a trend towards reduced SCR amplitudes relative to the control group.

All participants in this subgroup had scores in the normal range on the BFRIT. Moreover, the group effect remained a non-significant trend when the main ANCOVA was run again twice, with AED use ( $F(1, 26) = 4.06, p = .054$ ) and antidepressant use ( $F(1, 26) = 4.12, p = .053$ ), as additional between-group factors in the analysis. In these respective analyses, the effects of AED use ( $F(1, 26) = .472, p = .498$ ) and antidepressant use ( $F(1, 26) = .2, p = .659$ ) were not significant.

### **7.3.6. Exploratory analyses**

The exploratory correlational analyses focused on variables for which between-groups differences had been observed in the analyses described above.

### Recognition accuracy

Statistics for the correlations that were significant at the  $p < .01$  level in the DS group are presented in Table 34, in addition to the relevant statistics for control participants. Recognition accuracy scores for neutral faces were negatively correlated with IASC Abandonment Concerns (AC) and TEC total scores. These correlations were not significant in the control group. The relationship between accuracy for neutral faces and IASC AC scores remained significant after controlling for YoE ( $r = -.462$ ,  $p = .007$ ), as did the correlation with TEC total scores ( $r = -.456$ ,  $p = .008$ ).

**Table 34. Significant correlations ( $p < .01$ ) for facial expression recognition accuracy scores**

|                                | DS |       |         | Control |       |         |
|--------------------------------|----|-------|---------|---------|-------|---------|
|                                | n  | r/rho | p-value | n       | r/rho | p-value |
| <b>Neutral</b>                 |    |       |         |         |       |         |
| IASC                           |    |       |         |         |       |         |
| <i>Abandonment Concerns*</i>   | 36 | -.493 | .002    | 41      | .275  | .275    |
| TEC <i>total*</i>              | 37 | -.486 | .002    | 41      | .168  | .293    |
| <b>Fear</b>                    |    |       |         |         |       |         |
| IASC                           |    |       |         |         |       |         |
| <i>Affect Dysregulation</i>    | 36 | .448  | .006    | 41      | .064  | .690    |
| <b>Happiness</b>               |    |       |         |         |       |         |
| Emotional Stroop               |    |       |         |         |       |         |
| <i>Attentional bias scores</i> |    |       |         |         |       |         |
| <i>(Happy)</i>                 | 36 | -.429 | .009    | 41      | .176  | .270    |
| <i>Attentional bias scores</i> |    |       |         |         |       |         |
| <i>(Angry)*</i>                | 36 | -.452 | .006    | 41      | -.220 | .166    |

DS = dissociative seizures; IASC = Inventory of Altered Self-Capacities; FES = Family Environment Scale; TEC = Traumatic Experiences Checklist

\* denotes correlations that remained significant after controlling for YoE with partial correlations ( $p < .01$ )

Furthermore, a positive correlation between recognition accuracy scores for fearful faces and IASC Affect Dysregulation scores was observed in the DS group, but not in the control group. However, this relationship did not remain significant at  $p < .01$

after YoE was taken into account ( $r = .366$ ,  $p = .036$ ). Accuracy for recognition of happiness was negatively correlated with AB scores for angry and happy facial expressions on the emotional Stroop test (see Chapter 6). After controlling for YoE, the correlation with happy attentional bias scores no longer met the alpha level adopted here ( $r = -.344$ ,  $p = .05$ ), whereas the correlation with angry AB scores did ( $r = -.57$ ,  $p = .001$ ).

### SCRs

Table 35 displays the statistics for the SCR amplitude correlations that were significant in the DS group. There was a highly significant negative correlation between SCR amplitude for happy expressions and ictal mental state symptoms (most recent seizure). When YoE was controlled for using a partial correlation, this relationship was no longer significant at the required alpha level ( $r = -.670$ ,  $p = .012$ ). There was also a highly significant negative correlation between SCR amplitude for neutral faces and TEC mean impact scores. This correlation was not significant in the control group. However, after controlling for YoE, the relationship was no longer significant ( $r = -.451$ ,  $p = .122$ ) in the DS group either.

**Table 35. Significant correlations ( $p < .01$ ) for SCR amplitude scores (facial expression recognition task)**

|                                        | DS |       |         | Control |       |         |
|----------------------------------------|----|-------|---------|---------|-------|---------|
|                                        | n  | r/rho | p-value | n       | r/rho | p-value |
| <b>Happiness</b>                       |    |       |         |         |       |         |
| Ictal symptoms – mental state (recent) | 14 | -.674 | .008    |         |       |         |
| <b>Neutral</b>                         |    |       |         |         |       |         |
| TEC mean impact                        | 16 | -.645 | .007    | 15      | -.367 | .178    |

TEC = Traumatic Experiences Checklist; DS = dissociative seizures

\* denotes correlations that remained significant after controlling for YoE with partial correlations ( $p < .01$ )

### ***Differences between autonomic responders and non-responders***

As a means of exploring possible differences between autonomic responders and non-responders, exploratory analyses (t-tests, Mann-Whitney, chi-square tests) were

carried out to assess possible differences in relevant demographic characteristics (e.g. age, gender), psychosocial variables (e.g. trauma scores, dissociation, borderline personality features), and clinical details in the DS group (e.g. medication use, ictal symptoms, duration of disorder). Again, only p-values of less than .01 were considered significant in these analyses. None of the tests provided values that reached this significance level in either group.

## **7.4. Discussion**

### **7.4.1. Summary and interpretation**

The present study sought to extend previous research by experimentally testing explicit, conscious processing of emotional facial expressions in patients with DS, whilst simultaneously measuring SCRs elicited by these stimuli. Moreover, exploratory analyses were carried out to identify whether there were any relationships between the dependent measures in this study and relevant psychosocial variables (Chapter 5) or AB scores (Chapter 6). In addition, possible relationships between seizure-related variables and the dependent measures in the current study were examined.

#### ***Subjective responses***

The significant effect of expression on accuracy scores showed that accuracy varied significantly by expression type; however, the lack of a significant interaction between group and facial expression type suggested that this pattern was consistent between groups. The finding that happiness was most easily recognised is in accordance with other studies, which have shown that happiness is generally well-recognised (e.g. Hall & Matsumoto, 2004) and agreed upon in cross-cultural studies (e.g. Biehl et al., 1997). In contrast, neutral facial expressions were least accurately recognised in the current study. Again, this finding might be expected, as neutral expressions are inherently more ambiguous than expressions of specific emotions.

The between-groups difference in accuracy scores suggested that individuals diagnosed with DS are less accurate than healthy control participants in identifying

the emotional meaning of others' facial expressions. This finding was independent of the possible influence of depression, anxiety, educational level, medication and general face processing abilities. As such, it provides relatively robust evidence of facial expression processing difficulties in this population, thus supporting Hypothesis 1. However, the expected specificity for deficits in recognising negative facial expressions was not supported by this study. Instead, the findings suggested that recognition accuracy was affected across the range of facial expressions, including happiness and neutrality. The lack of significant group effects (or interactions) for the perception of emotional intensity in facial expressions indicated that this sample of individuals with DS were generally accurate in assessing the extent/level of emotional arousal displayed facially. This finding does not support Hypothesis 2, in which it was predicted that patients with DS would perceive elevated levels of emotional intensity in facial expressions.

The observed deficit in facial expression recognition is similar to that observed in patients with BPD, depression, depersonalisation disorder and PTSD, although the impairments have often been restricted to negative and/or neutral expressions in those groups. Nevertheless, the results suggest that patients with DS misinterpret others' emotional states to a greater extent than controls, and so it is possible that this might negatively affect patients' relationships and daily interactions. For example, instances in which another person is experiencing a neutral or positive emotional state could be misinterpreted as reflecting some variation of negative emotion, or different intense negative emotional expressions could be confused by patients.

The negative correlation between recognition accuracy for neutral facial expressions and abandonment concerns indicates that misinterpreting these signals is linked to maladaptive relationship schemata. Furthermore, the finding that reduced recognition accuracy for neutral faces was associated with higher TEC total scores suggests that, in patients with DS, difficulties in identifying neutrality in facial expressions might somehow be related to adverse/traumatic life experiences. It is possible that traumatic experiences involving interpersonal relationships (e.g. abuse) might be associated with a lack of consistency between others' facial expressions and their behaviour, or between others' facial expressions and the emotional consequences for

the individual viewing the expression (e.g. a neutral face in an abuser could be associated with abusive behaviour and/or intense negative affect in the victim). If such experiences occurred during development, they might result in facial expressions being perceived as unpredictable and inconsistent, or as signalling threat when none is present. Interestingly, facial expression recognition deficits in individuals with BPD have been reported to be associated with childhood trauma (i.e. physical and emotional abuse; Nicol et al., 2014).

The negative correlation between recognition of happy faces and preconscious attentional bias towards angry faces on the emotional Stroop test suggests that a greater tendency to perceive negative emotions or neutrality in happy faces is associated with preconscious hypervigilance for angry faces. Combined, these tendencies might lead patients with DS to perceive more hostility and less social acceptance or positive regard in other people. If this were the case, it would be likely to contribute to elevated emotional distress and relationship difficulties. Further research is required to explore the ways in which preconscious and conscious processing of social emotional signals are related in patients with DS.

### ***Autonomic responses***

The findings relating to SCRs did not support Hypothesis 3, which predicted heightened levels of autonomic responding to the facial expression stimuli. There were no group differences in the frequency of positive SCRs or the proportion of each group showing such responses across the facial expression types. Moreover, the amplitude data indicated that for those participants classified as ‘responders’, those with DS showed reduced SCR amplitudes relative to controls. This finding is similar to that observed in depersonalisation disorder, in which reduced autonomic responding has been observed for facial expressions of disgust (Sierra et al., 2006). However, in the current study, patients with DS were shown to have reduced SCR amplitudes across facial expression types (including neutral), rather than for any one specific expression.

The SCR findings indicate that, as a group, patients with DS are equally likely to exhibit autonomic responses to facial expressions as healthy controls; however, those

patients who are consistently responsive to such stimuli show an overall reduction in the extent of these responses. The findings indicate that when emotional facial expressions are available for conscious processing and attention is explicitly directed at them, a subgroup of patients with DS show inhibited autonomic responding to these, and that this inhibition is not emotion-specific.

It could be the case that this reduced autonomic responding reflects some form of habituation or desensitisation to the facial expressions. Repeated previous experience of perceived or actual negative facial expressions could lead to a dampening of autonomic responses to these. On the other hand, it could be part of a dissociative response to these potentially threatening or distressing social cues. Skin conductance changes are relatively slow responses, and so, they may well occur after conscious awareness of the stimuli has been achieved. In this particular disorder, whilst hypervigilance for emotional facial expressions might occur at a preconscious level, conscious awareness of the stimuli may be associated with rapid inhibition of emotional responses to them, thus leading to abnormally low SCR amplitudes. Ontogenetically, this mechanism might develop as a means of automatically detaching or distancing the individual from the physiological and subjective experience of emotional responding to the stimuli. As yet, these are tentative hypotheses.

#### **7.4.2. Strengths and limitations**

As with previous chapters, only the strengths and limitations specific to the current chapter are presented in this section. An important strength of the study was the collection of both subjective and autonomic measures of emotional responding to consciously perceived facial expression stimuli. This the first study to experimentally examine conscious (explicit) facial expression processing in this clinical group, and the inclusion of the SCR measure allowed a direct comparison of conscious and more automatic responding to such stimuli, in the same participants.

The findings have provided an important insight into the possibility that patients with DS may experience reduced autonomic arousal in response to facial expressions, coinciding with difficulties in consciously differentiating their emotional meaning. The present study also included measures of general face processing, intellectual

functioning, and depression/anxiety, and this allowed a closer examination of the possible influence of these variables on the measures of emotional responding. In the present study, none of these variables (or medication effects) sufficiently accounted for the observed reduction in subjective recognition or autonomic arousal, in response to the facial expressions.

Another strength of the study included the use of well-known standardised facial expression stimuli, which have been found to elicit high levels of agreement cross-culturally. These stimuli have been used widely in similar research and, therefore, improve comparability with other studies and allow more precise replicability of the current paradigm. One possible limitation of these stimuli is that the ethnicity of the actors is limited (all actors are Caucasian), and studies have shown that recognition of facial expressions can be affected by the ethnicity of the model and that of the respondent (Elfenbein & Ambady, 2002). Future studies might seek to utilise more ethnically diverse stimuli sets. Nonetheless, as the groups in the present study were reasonably well-matched in terms of ethnicity, this factor is unlikely to have been influential in the current study.

Another possible limitation relating to the facial stimuli is that some were included in both the experiment described in Chapter 6 and that described in the current chapter. The stimulus set includes a limited number of actors, some of whom did not pose all facial expressions (i.e. fear). Given that the experiment described in Chapter 6 required ten different actors (five male and five female), and the present study required six actors (three male, three female) and an additional actor for the practice items, it was not possible to avoid some duplication of stimuli between the tasks. Moreover, the stimuli (actors) were selected on the basis of the normative ratings, as the best exemplars of the emotions being displayed (i.e. having received the highest normative ratings). Moreover, the stimuli were coloured and presented subliminally in the emotional Stroop test, whereas they were grey-scale and presented supraliminally in the current experiment. Nevertheless, it is a possibility that prior exposure to some of the facial stimuli might have influenced the findings reported here. Nonetheless, as both groups underwent the same order of administration of



the computerised tests, any influence of prior exposure should have been consistent between groups.

Due to the length of the experiment (necessitated by the ITI required for SCR recording), it was necessary to restrict the number of conditions included, which could be viewed as a limitation. Only one positive condition (happiness) was included, whereas three negative conditions (anger, disgust, fear) were examined, along with neutrality. It could be argued that the group effect provides stronger evidence for impaired recognition of negative expressions, due to the combined possible influence of three negative conditions. Future studies might seek to balance the number of positive and/or reducing the number of negative conditions.

Moreover, the inclusion of neutral facial expressions as an additional condition in the current study is another issue to consider. In the current study, the neutral condition was included as an experimental condition as it was thought to be of interest to examine patients' responses to such ambiguous expressions. Whilst some studies include neutral expressions as a control condition only (i.e. as a comparison condition), neutral expressions may be processed differently in some psychological disorders and so might not represent a genuine (unemotional) control condition. For example, there is some evidence to suggest that patients with BPD (see Daros et al., 2013), somatoform disorders (Pollatos et al., 2011) and depression (e.g. Leppänen et al., 2004) tend to show altered recognition of neutral facial expressions compared to control groups. In the current study, the significant expression effect combined with the lack of interaction between group and expression (on accuracy scores) did not suggest that the DS group were specifically impaired in recognising neutral expressions, but rather that both groups found these expressions the most difficult to distinguish from the other expressions.

Regarding SCR measurement and analysis, this study followed generally accepted standards in most respects (i.e. Boucsein, 1992; Dawson et al., 2000; Lykken & Venables, 1971). However, the apparatus included in the present study included the use of dry (stainless steel) electrodes, recommended by the manufacturer of the psychophysiological recording equipment (ADInstruments). In contrast, the generally

accepted recommendations (see Boucsein et al., 2012) refer to the use of silver-silver chloride (Ag/AgCl) electrodes with an electrolyte paste or gel. Nonetheless, as both groups' SCRs were recorded with the same electrodes in the current study, the use of the dry electrodes is not likely to explain the group differences observed. Future studies might seek to replicate the present findings using the recommended electrode type. Moreover, the inclusion of other psychophysiological measures (e.g. electrocardiogram, electroencephalography) could be included in further studies of facial expression processing in this group. The use of functional neuroimaging techniques to examine the correlates of central nervous system activity during similar tasks is also an important direction for additional studies.

#### **7.4.3. Conclusions**

Together, the findings of the present study are suggestive of a reduction in psychophysiological and subjective responding to emotional facial expressions in patients with DS, relative to healthy individuals. This pattern of responding may have become a habitual way of responding to others' emotional expressions within the context of significant traumatic life events. It is possible that reduced subjective and somatic responding to others' facial expressions could represent a habituated response to others' emotional cues. On the other hand, it could be regarded as a dissociative response that allows the individual to remain detached from potentially threatening or distressing social signals, or to avoid conscious awareness of their meaning altogether. Either way, it is likely that reduced recognition and autonomic responding to facial expressions may lead to difficulties in interpersonal functioning and elevate levels of emotional distress. Therefore, this emotional response style could serve as a predisposing factor for the development of DS, but might also contribute to triggering and maintaining the occurrence of DS on an ongoing basis.

## **Chapter 8. An experimental study of subjective and autonomic responsivity to affective pictures in patients diagnosed with dissociative seizures**

### **8.1. Introduction**

#### **8.1.1. Background**

The findings presented in previous chapters suggest that there may be important differences in how patients with DS process and respond to emotional facial expressions, relative to healthy control participants. However, the possibility remains that patients with DS may also differ in their responses to other emotional stimuli, beyond those purely in the social domain. It was, therefore, considered to be of interest to examine affective responses to more general emotional stimuli, including those representing different types of emotionally significant objects or scenes. The aim of this study was to assess the same subjective and autonomic response domains as in Chapter 7, but in relation to more general affective stimuli. Images from the International Affective Picture System (IAPS; Lang et al., 2005) were selected for this purpose, being the most widely used standardised stimuli in this area.

The IAPS images (Lang et al., 2005) provide emotion researchers with a standardised set of stimuli with which to conduct readily replicable and comparable studies. The images vary considerably, depicting a wide range of scenes, objects and people. The scenes also vary on three affective dimensions: valence (positive, negative, neutral), level of arousal (low to high) and 'dominance' (control). The availability of detailed normative data, based on large samples of adults and children, allows the investigator to select appropriate images on the basis of normative ratings of the three affective dimensions. The most commonly used ratings in published research are the valence and arousal scales. The IAPS images have now been utilised widely in research on emotional processing in healthy and clinical populations, including studies of subjective, psychophysiological and neural responses to such stimuli.

Some investigators have examined responsivity to IAPS images in individuals diagnosed with psychological disorders that are related to DS, such as borderline personality disorder (BPD). For example, Herpertz and colleagues (1999) reported that their sample of patients with BPD showed reduced SCR magnitude for all three valence categories of IAPS stimuli, relative to healthy controls. This study was suggestive of inhibited autonomic responding to these stimuli in that group. However, Marissen, Meuleman, and Franken (2010) observed greater event-related potentials for negative IAPS stimuli in patients with BPD relative to a healthy control group. Feliu-Soler et al. (2013) more recently reported comparable physiological (salivary cortisol) and subjective (SAM) emotional responses to negative IAPS stimuli in a larger sample of BPD patients relative to healthy controls.

More recent research indicates that patients with BPD may show altered subjective responses (increased arousal, greater negativity) for particular types of visual images, such as those suggestive of sexual abuse (Sauer et al., 2014). This highlights the fact that such patients may respond differentially to stimuli that are of potential aetiological relevance to their disorder, or to the symptoms and schemata present. However, an important caveat is that these findings did not survive statistical correction for depressive symptoms and the stimuli included were not standardised IAPS images, but instead consisted of images sourced from the internet. Nonetheless, this raises the importance of careful selection of affective images in this type of study, with consideration of the extent to which particular types of images could be of immediate relevance to the patient group being studied. It also points to the important decision of whether or not to statistically control for possibly confounding psychological symptoms, such as depression or anxiety. Together, the findings in patients with BPD suggest that physiological and subjective responsivity to IAPS stimuli may vary with the particular measures obtained and with the stimuli selected. More research is needed to further clarify subjective and psychophysiological response tendencies to IAPS stimuli in individuals with BPD.

There are a variety of other methodological issues to be considered when interpreting the results of studies such as those discussed above. For example, it is known that symptoms of state dissociation can influence dependent variables when

studying groups who tend to experience elevated dissociative symptoms such as those with BPD or PTSD (e.g. Ebner-Primer et al., 2005); therefore, measuring and taking into account such symptoms is important. Moreover, the treatment history of patients included is also a factor. As with many samples recruited within mental health services, possible medication effects need be considered. In addition, the majority of BPD patients included in the study of Herpertz et al. (1999) had undergone an intensive inpatient treatment programme, which may well have included emotion regulation training, and the development of coping skills, for example. Such skills may have allowed the patients to regulate their responding to a greater extent than control participants, who were documented to have had no psychiatric treatment in their histories. Moreover, gender is also a consideration in recruiting participants. For example, Herpertz et al. (1999) restricted their sample to female patients only, which obviously limits the ability to generalise the results to male BPD patients.

### **8.1.2. Rationale, aims & hypotheses**

When the present experiment was devised, there had been no experimental studies of affective picture processing in patients with DS. However, one study of this nature was published during data collection for the present thesis. As described in Chapter 3 (section 3.5.2), Roberts and colleagues (2012) measured subjective (valence, arousal), behavioural (facial, bodily), and autonomic (heart rate, heart rate variability) responses to IAPS stimuli in patients with DS. These stimuli were presented within a paradigm that allowed conscious and explicit processing of the stimuli. The DS group were similar to healthy controls in the valence of their emotional responses to the images, but they reported more emotional arousal in response to the neutral and positive images than the control group.

The elevations in arousal ratings in the DS group were discussed in relation to the tendency to somatise, the authors suggesting that patients with DS may experience emotion in primarily visceral ways. However, the investigators did not examine whether somatisation scores correlated with or predicted the intensity ratings in the DS group. Moreover, reduced respiratory sinus arrhythmia (an indicator of heart rate variability) at baseline was also reported in the DS group, interpreted as evidence

for an underlying vulnerability to affective dysregulation. Heart rate variability differences in patients with DS have also been reported by other authors (see Chapter 2, section 2.1.5). The general strengths and weaknesses of this study were discussed in Chapter 3. Whilst the findings are highly relevant to the current study, the findings did not contribute to the aims, rationale or hypotheses of the present research.

The current experiment was designed with the aim of assessing subjective and autonomic responding to IAPS stimuli. It was intended that this study would provide an insight into any differences between patients with DS and healthy controls, in subjective emotional responses to general affective stimuli. Previous studies had not examined such responses, at the time the research was designed. Moreover, the study aimed to assess whether autonomic responding to these stimuli differed between patients with DS and healthy controls. An additional aim was to carry out exploratory analyses to assess how seizure-related variables (i.e. seizure frequency, ictal symptoms) and psychosocial factors (i.e. trauma, dissociation, BPD features) might relate to any observed differences in affective responding.

On the basis of the available literature and the model presented in Chapter 3 (section 3.6), the following hypotheses were tested:

1. Patients with DS were predicted to report more negative ratings for the affective pictures, relative to the healthy control group. It was expected that these differences would be apparent for negative images only.
2. Patients with DS were predicted to display elevated ratings of arousal for the affective pictures, relative to the healthy control group. It was expected that these differences would be apparent for negative images only.
3. Higher levels of autonomic responding (i.e. more frequent and higher amplitude of SCRs) were predicted to be observed in patients with DS, relative to the healthy control group. These differences were expected to be observed for negative images only.

In order to test these hypotheses, an experiment was devised that was as similar in structure as possible to that described in Chapter 7. The difference in the current experiment was the use of the IAPS stimuli, instead of facial expressions, and the use of different subjective response scales (section 8.2.2). In the present study, the dependent variables were subjective ratings (arousal, valence) and autonomic responses (SCRs). The responses of the DS sample were compared to those of a healthy control group.

## **8.2. Methodology**

After initial piloting of the task, it was clear that the task was too long. Therefore, the number of stimuli in each condition was reduced from eight to six, and the ITI was reduced from 25 seconds to 15 seconds. No other changes were made to the experiment after piloting. The final design is reported below.

### **8.2.1. Participants**

Details of recruitment procedures and inclusion criteria for this study were described in Chapter 4 (sections 4.2.1. and 4.2.2). The final sample for this study included 42 control participants and 39 patients diagnosed with DS. The two participants who did not complete the task had asked to refrain from doing so, due to fatigue during the testing session.

### **8.2.2. Materials**

#### ***Experimental materials***

The hardware and software used to acquire subjective responses in this task were identical to those described in the previous chapter (section 7.2.2). However, the experimental stimuli used in the current task were taken from the IAPS set (Lang et al., 2005). On the basis of the normative arousal and valence ratings provided with this set, stimuli were selected from each of the following categories: positive/high arousal, positive/low arousal, neutral, negative/high arousal, negative/low arousal.

Six pictures were chosen from each category with consideration given to several issues. Pictures with very extreme and graphic content were excluded; these included items depicting severe mutilations, frontal nudity, direct interpersonal violence and explicit sexual scenes. Pictures were selected that were maximally representative of their category (in terms of normative scores) but that were also considered appropriate for the testing situation. It was also intended to include as much variety of content as possible within each condition. A list of all of the pictures used in the study can be found in Appendix 17, with examples of each condition. In order to assist participants in making their subjective responses to the stimuli, two digitised versions of the Self-Assessment Manikin (SAM) were used (arousal, valence), each with a nine-point scale. The SAM displays can be found in Appendix 18.

### ***Skin conductance recording apparatus***

All apparatus used to acquire and analyse the SCR data was identical to those described in Chapter 7 (section 7.2.2).

### ***Neuropsychological measures***

*Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999)*

See Chapter 6 (section 6.2.2) for a discussion of the inclusion of this test.

*Visual Object & Space Perception Battery (VOSP; Warrington & James, 1991)*

One subtest (Object Decision, OD) of the VOSP was included to assess general object perception/recognition. This test was used to measure whether there were any between-groups differences in perception of visual stimuli. Each page of the test includes four black shapes presented on a white background. Three of the shapes are nonsense shapes that do not represent actual objects, whereas one shape does constitute the silhouette of a real object. Participants are requested to select the real object on each page. The VOSP OD subscale has clinical cut-off scores of 15 (participants aged <50 years) and 14 (individuals aged >50 years), below which a significant impairment is indicated.



*Wechsler Memory Scale – Third Edition (WMS-III; Wechsler, 1997)*

The Family Pictures I subscale was administered to measure immediate memory for complex visual scenes (recall). The use of the Family Pictures I subtest is relevant to the current chapter as it examines participants' ability to retain complex visual scenes in short-term memory. The experimental task described presently requires participants to retain scenes of varying complexity in short-term memory, in order that they can provide their subjective responses during the ITI. Therefore, controlling for short-term memory for such stimuli is important, to ensure that no between-groups differences in memory ability were responsible for any differences on the task itself. The raw scores on the Family Pictures I subtest can be transformed into scaled scores on the basis of age, based on normative data.

### **8.2.3. Design**

The experiment was a mixed factorial design with one between-groups factor (diagnostic status: DS, control) and one within-groups factor (emotional category: neutral, negative/high arousal, negative/low arousal, positive/high arousal, positive/low arousal). The dependent variables were subjective ratings of valence (0-9, negative-positive), arousal (0-9, high-low) and SCRs. Several measures of SCR were examined, including amplitude and response frequency. These measures were outlined in the previous chapter (section 7.2.5). There were six stimuli in each emotional category, yielding 30 experimental trials. These stimuli were presented in a new pseudo-random order for every participant, with no more than two stimuli from the same condition presented consecutively.

### **8.2.4. Procedure**

Prior to commencing the experimental task, all participants underwent a five-minute habituation period with the physiological sensors attached. Following this, the research student went through the standardised instructions with the participant (Appendix I9). These instructions were based on those provided in the IAPS manual (Lang et al., 2005), with some modifications. The instructions were presented in white font (size 16-18, Tahoma style) against a black background on the computer screen. Any questions that arose were answered at this time.

The task began with three practice trials, with three stimuli that were normatively rated as low in arousal. Participants practised rating these stimuli before proceeding to the main task. The experimental trials consisted of a 15-second presentation of a central white fixation cross, against a black background. This ITI was selected due to the requirements of the SCR recording. After the fixation cross, the target stimuli were presented on the screen for six seconds as described by M.M. Bradley et al. (2001). Immediately after stimulus offset, the two rating screens were presented consecutively. The order of the two screens was randomised for each participant. Participants were instructed to make their ratings using the number keys. On completion of the ratings, the ITI (fixation) commenced again before the next stimulus was presented. On completion of all trials, a screen appeared with a thank-you message. Any remaining questions were answered and the participant's wellbeing was also checked.

#### **8.2.5. SCR data acquisition, extraction & reduction**

All methods for acquiring, extracting and reducing the SCR variables were as described in the previous chapter (see section 7.2.5.).

#### **8.2.6. Statistical analysis**

##### ***Subjective measures***

Mean ratings on the arousal and valence scales (0-9) were entered as the dependent variables in two mixed factorial ANCOVAs with group (DS, control) as the between-subjects factor and condition (neutral, negative/high arousal, negative/low arousal, positive/high arousal, positive/low arousal) as the within-subjects factor. Covariates for these analyses are detailed in the relevant sections below.

##### ***Skin conductance responses***

SCR amplitude data were analysed separately with mixed factorial ANCOVAs as described for the subjective ratings. The percentage of trials in which positive responses were observed were also entered as the dependent variable in a similarly constructed analysis. Chi-square tests were used to assess the relative proportion of each group showing at least one positive SCR for each condition.

### ***Exploratory analyses***

The exploratory analyses proceeded as described in Chapter 7 (section 7.2.6). The same psychosocial and seizure-related variables were included as described in that section. In addition, the methods used were similar to those described in the previous chapter. Only the dependent variables found to differ significantly between-groups in the present study were explored in these analyses.

## **8.3. Results**

### **8.3.1. Participant characteristics**

Forty-two control participants and 39 patients with DS completed the experiment. Demographic characteristics of the included sample are shown in Table 36. There were no significant group differences in age, gender, ethnicity, handedness, marital status, smoking, and socio-economic status. However, the control group reported significantly more years of education (YoE) than the DS group and there was a non-significant trend for control participants to be more likely to have completed further/higher education. YoE was, therefore, entered as a covariate in subsequent analyses.

Patients with DS ( $n = 28$ , 71.8%) were more likely to be taking prescribed medication than the control group ( $n = 10$ , 23.8%;  $\chi^2(1, 81) = 18.7$ ,  $p < .001$ ). Moreover, a higher proportion of the DS group ( $n = 22$ , 56.4%) reported the presence of a current medical diagnosis ( $\chi^2(1, 81) = 15.9$ ,  $p < .001$ ) than in the control group ( $n = 6$ , 14.3%). Fourteen patients with DS (35.9%) were taking anti-epileptic drugs (AEDs), most commonly sodium valproate, carbamazepine, pregabalin and levetiracetam. Fifteen patients (38.5%) with DS were taking anti-depressant medications, most commonly selective serotonin reuptake inhibitors (SSRIs). The most common SSRIs reported were fluoxetine ( $n = 6$ ), citalopram ( $n = 3$ ) and sertraline ( $n = 3$ ).

In the DS group, the average (median) length of time since seizure onset was 60 months (range = 9-432; interquartile range = 90). The median reported seizure frequency was 4 per month (range = 0-274; interquartile range = 14).

**Table 36. Demographic characteristics (by group) for participants completing the affective picture experiment**

|                               | <b>DS<br/>(n = 39)</b>                                               | <b>Control<br/>(n = 42)</b>                                         | <b>Test<br/>statistics</b>          |
|-------------------------------|----------------------------------------------------------------------|---------------------------------------------------------------------|-------------------------------------|
| Age in years<br>(Mean, SD)    | 37.9 (13.2)                                                          | 37.3 (11.8)                                                         | t (79) = -.212, p = .832            |
| Gender                        | Male = 8 (20.5%)<br>Female = 31 (79.5%)                              | Male = 7 (16.7%)<br>Female = 35 (83.3%)                             | $\chi^2$ (1, n=81) = .198, p = .656 |
| Handedness                    | Right = 29 (74.4%)<br>Left / ambidextrous = 10 (25.6%)               | Right = 37 (88.1%)<br>Left / ambidextrous = 5 (11.9%)               | $\chi^2$ (1, n=81) = 2.53, p = .112 |
| Ethnicity                     | White = 31 (79.5%)<br>Non-white = 8 (20.5%)                          | White = 28 (66.7%)<br>Non-white = 14 (33.3%)                        | $\chi^2$ (1, n=81) = 1.68, p = .195 |
| YoE                           |                                                                      |                                                                     |                                     |
| Mean (SD)                     | 12.9 (2.5)                                                           | 14.1 (2.6)                                                          | U (81) = 600, p = .036              |
| Median (IQR)                  | 13 (3)                                                               | 14.5 (4.3)                                                          |                                     |
| Qualifications                | GCSEs / none = 15 (38.5%)<br>Further / higher = 24 (61.5%)           | GCSEs / none = 8 (19%)<br>Further / higher = 34 (81%)               | $\chi^2$ (1, n=81) = 3.75, p = .053 |
| Marital status                | Currently single = 24 (61.5%)<br>Long-term relationship = 15 (38.5%) | Currently single = 33 (78.6%)<br>Long-term relationship = 9 (21.4%) | $\chi^2$ (1, n=81) = 2.81, p = .093 |
| Socio-economic status (NSSEC) | 1 = 18 (46.2%)<br>2,3,4 or 5 = 21 (53.8%)                            | 1 = 18 (42.9%)<br>2,3,4 or 5 = 24 (57.1%)                           | $\chi^2$ (1, n=81) = .089, p = .765 |
| Smoking                       | Yes = 10 (25.6%)<br>No = 29 (74.4%)                                  | Yes = 9 (21.4%)<br>No = 33 (78.6%)                                  | $\chi^2$ (1, n=81) = .2, p = .655   |

SD = standard deviation  
IQR = interquartile range  
YoE: years of full-time education (or equiv.)  
DS = dissociative seizures

NSSEC: National Statistics Socio-economic Classification system  
1 = Higher managerial, administrative and professional occupations  
2 = Intermediate occupations  
3 = Small employers and own account workers  
4 = Lower supervisory and technical occupations  
5 = Semi-routine and routine occupations

### 8.3.2. Neuropsychological measures

The neuropsychological findings are displayed in Table 37. There were no significant differences in full-scale IQ, Vocabulary or Matrix Reasoning scores between groups. A significant between-groups difference was observed on the VOSP OD subscale, with the DS group performing significantly better than the control group. Inspection of z-scores indicated that one outlier was present in the control group; however, a significant difference remained after removing this participant's score. Furthermore, the significant difference remained after removal of any participants scoring below the cut-off on the test.

**Table 37. Neuropsychological findings (affective picture task)**

|                                          | <b>DS</b>    | <b>Control</b> | <b>Test statistic (df)</b> | <b>p-value</b> |
|------------------------------------------|--------------|----------------|----------------------------|----------------|
| <b>WASI</b>                              | n = 39       | n = 42         |                            |                |
| FSIQ (Mean, SD)                          | 103.8 (14.6) | 108.2 (13.3)   | t (79) = 1.4               | p = .165       |
| Vocabulary T scores (Mean, SD)           | 51.7 (11.2)  | 55.4 (9.9)     | t (79) = 1.5               | p = .125       |
| Matrix Reasoning T scores (Median, IQR)  | 54 (10)      | 55.5 (15)      | U (81) = 725.5             | p = .376       |
| <b>VOSP OD</b>                           | n = 38       | n = 42         | U (80) = 551.5             | p = .016       |
| Median (IQR)                             | 18 (3)       | 17 (3)         |                            |                |
| <b>WMS-III</b>                           | n = 38       | n = 42         |                            |                |
| Family Pictures scaled scores (Mean, SD) | 8.7 (2.4)    | 7.3 (1.9)      | t (78) = -2.99             | p = .004       |
| Visual Immediate (Mean, SD)              | 98.7 (14.2)  | 94.9 (12.4)    | t (78) = -1.26             | p = .213       |

DS = dissociative seizures; SD = standard deviation; df = degrees of freedom; IQR = interquartile range;  
 WASI = Wechsler Abbreviated Scale of Intelligence; FSIQ = Full-scale Intelligence Quotient;  
 VOSP OD = Visual Object & Space Perception Battery – Object Decision subscale;  
 WMS-III = Wechsler Memory Scale – Third Edition

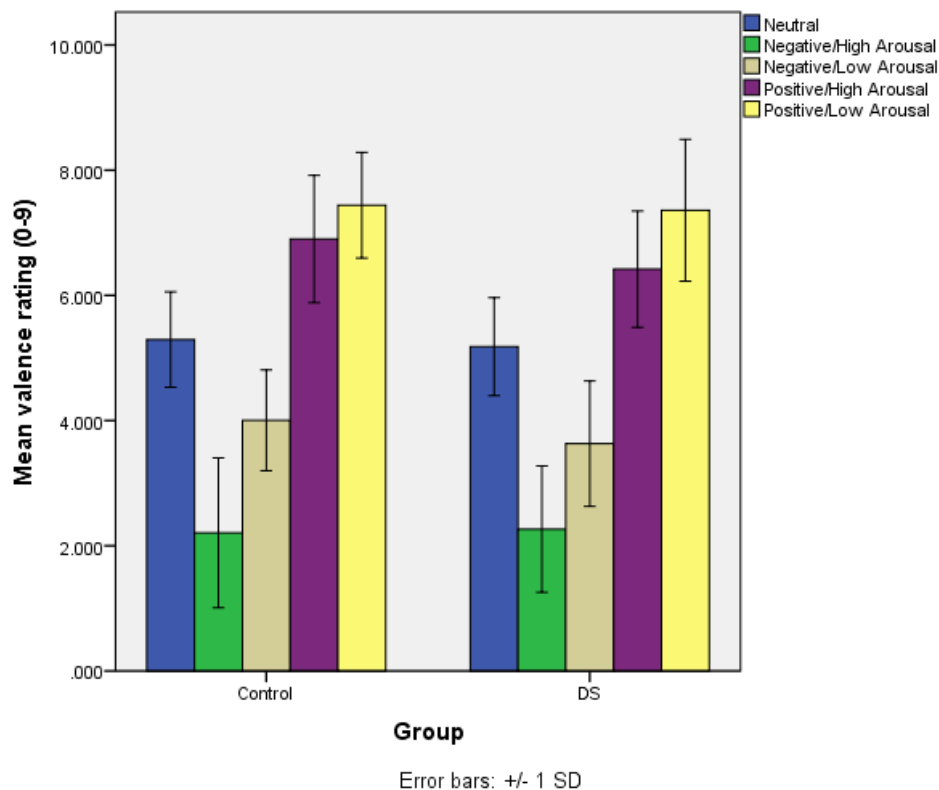
There was no overall between-groups difference on the Visual Immediate Index (including Faces I and Family Pictures I scores). However, there was a significant difference on the Family Pictures I subtest, with the DS group again performing better than the control group. Inspection of z-scores did not reveal any outliers on this subtest.

### 8.3.3. Subjective ratings

#### Valence

Figure 13 displays the descriptive statistics for the valence ratings. Due to the existence of several possible covariates for entry into the analysis (VOSP OD, WMS-III Family Pictures I, HADS Anxiety, HADS Depression, YoE), and the possibility of loss of statistical power if all were included, correlations between the possible covariates and valence ratings for each condition were examined, across the entire sample. YoE was not significantly correlated with valence ratings for any of the conditions; therefore, it was not entered into the analysis. Moreover, HADS Depression scores did not correlate significantly with valence ratings for any condition; therefore depression scores were also not entered into the model.

**Figure 13. Mean valence ratings by group and condition (IAPS task)**



The mixed factorial ANCOVA indicated that there was no main effect of group ( $F(1, 75) = .701, p = .405$ ) and no interaction between group and condition ( $F(2.38, 178.4) = .659, p = .544$ ). The only (marginally) significant covariate was anxiety ( $F(1, 75) = 3.95, p = .051$ ), which had a negative influence on valence ratings in all conditions except for positive low arousal images. There was a highly significant main effect of condition ( $F(2.38, 178.4) = 15.5, p < .001$ ), as might be expected. Pairwise comparisons indicated that all conditions differed significantly from each other, with the highest valence ratings for both positive conditions, and the lowest ratings for both negative conditions. The negative high arousal condition elicited the most negative ratings, and the positive low arousal condition the most positive.

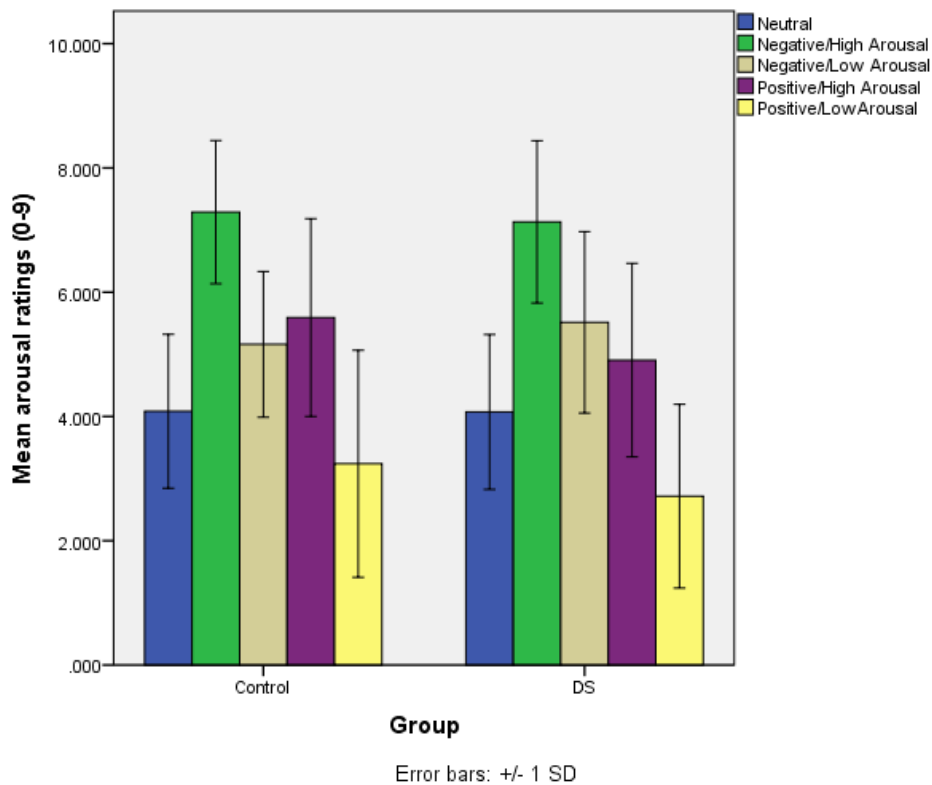
### ***Arousal***

Figure 14 displays the descriptive statistics for the arousal ratings. Correlational analyses indicated that VOSP OD scores were not significantly related to arousal ratings for any condition; therefore, VOSP OD was not entered as a covariate. Moreover HADS Depression scores were not entered as a covariate for the same reason. With the remaining covariates entered (YoE, HADS Anxiety, WMS-III Family Pictures I), the analysis revealed that there was no overall main effect of group ( $F(1, 75) = 1.14, p = .289$ ) and no interaction between group and condition ( $F(3.28, 246.2) = .371, p = .792$ ). None of the covariates were significant. However, there was a significant main effect of condition ( $F(3.28, 246.2) = 5.39, p = .001$ ). Pairwise comparisons showed that the negative high arousal condition elicited the highest arousal ratings, and the positive low arousal condition elicited lower arousal ratings than all other conditions.

#### **8.3.4. Skin conductance responses (SCRs)**

Table 38 shows the descriptive statistics for all SCR variables described below. SCR data for two of the control participants who completed the experiment were not usable due to technical failures.

**Figure 14. Arousal ratings by condition and group (IAPS task)**



### **Total trials included**

A mixed factorial ANOVA showed that, for the total number of trials retained for the SCR analysis, there was a marginally significant main effect of group ( $F(1, 78) = 3.84, p = .053$ ), with significantly more trials retained for the DS group (estimated marginal mean = 5.91; standard error = .061, 95% CI = 5.79, 6.03) than the controls (estimated marginal mean = 5.74, standard error = .059, 95% CI = 5.62, 5.86). On the other hand, there was no main effect of condition ( $F(3.84, 299.4) = .617, p = .644$ ), and no interaction between condition and group ( $F(3.84, 299.4) = .690, p = .594$ ). This finding suggested that there was less noise and artefact present on trials in the DS group than in the control group, indicating that patients moved less and had more steady breathing than controls during the experiment.

### **Percentage of positive responses**

Correlations indicated that, of the potential covariates, only HADS Anxiety ( $p = .024$ ) and Depression ( $p = .005$ ) scores correlated with the proportion of positive responses for at least one condition; therefore, both of these sets of scores were entered as covariates.



**Table 38. Skin conductance response (SCR) descriptive statistics (affective picture task)**

|                                                                                    | <b>n</b>                | <b>DS</b>                                                                                                                                                                                                                                                              | <b>Control</b>                                                                                                                                                                                                                                                             |
|------------------------------------------------------------------------------------|-------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Total number of trials retained for analysis (0-6) (Median, IQR) (Mean, SD)</b> | DS = 39<br>Control = 40 | Neutral: Mdn = 6 (0); Mean = 5.9 (.384)<br>Negative High: Mdn = 6 (0); Mean = 5.9 (.384)<br>Negative Low: Mdn = 6 (0); Mean = 5.9 (.339)<br>Positive High: Mdn = 6 (0); Mean = 6 (0)<br>Positive Low: Mdn = 6 (0); Mean = 5.9 (.339)                                   | Neutral: Mdn = 6 (0); Mean = 5.7 (.64)<br>Negative High: Mdn = 6 (0); Mean = 5.8 (.53)<br>Negative Low: Mdn = 6 (0); Mean = 5.8 (.577)<br>Positive High: Mdn = 6 (0); Mean = 5.7 (.716)<br>Positive Low: Mdn = 6 (0); Mean = 5.7 (.694)                                    |
| <b>Percentage of trials with positive SCRs (0-100%) (Median, IQR) (Mean, SD)</b>   | DS = 39<br>Control = 40 | Neutral: Mdn = 16.7 (50); Mean = 30.3 (29.3)<br>Negative High: Mdn = 33.3 (50); Mean = 35.5 (30.4)<br>Negative Low: Mdn = 33.3 (33.3); Mean = 38.5 (25.5)<br>Positive High: Mdn = 33.3 (50); Mean = 39.7 (26.7)<br>Positive Low: Mdn = 33.3 (66.7); Mean = 34.7 (29.1) | Neutral: Mdn = 33.3 (38.3); Mean = 34.2 (26.7)<br>Negative High: Mdn = 41.7 (50); Mean = 42.3 (28.1)<br>Negative Low: Mdn = 33.3 (33.3); Mean = 35.7 (23.9)<br>Positive High: Mdn = 33.3 (48.3); Mean = 38.6 (26.2)<br>Positive Low: Mdn = 33.3 (33.3); Mean = 38.4 (26.6) |
| <b>Percentage of participants displaying &gt;1 positive SCR n (% of group)</b>     | DS = 39<br>Control = 40 | Neutral: 27 (69.2%)<br>Negative High: 31 (79.5%)<br>Negative Low: 34 (87.2%)<br>Positive High: 34 (87.2%)<br>Positive Low: 29 (74.4%)                                                                                                                                  | Neutral: 34 (85%)<br>Negative High: 36 (90%)<br>Negative Low: 35 (87.5%)<br>Positive High: 34 (85%)<br>Positive Low: 35 (87.5%)                                                                                                                                            |
| <b>SCR amplitude in microSiemens (Median, IQR) (Mean, SD)</b>                      | DS = 20<br>Control = 23 | Neutral: Mdn = .291 (.83); Mean = .604 (.83)<br>Negative High: Mdn = .762 (.91); Mean = .845 (.733)<br>Negative Low: Mdn = .434 (.54); Mean = .539 (.44)<br>Positive High: Mdn = .609 (.81); Mean = .903 (1.17)<br>Positive Low: Mdn = .527 (.82); Mean = .649 (.595)  | Neutral: Mdn = .296 (.56); Mean = .417 (.45)<br>Negative High: Mdn = .63 (.94); Mean = .657 (.624)<br>Negative Low: Mdn = .456 (.77); Mean = .512 (.443)<br>Positive High: Mdn = .416 (.57); Mean = .433 (.417)<br>Positive Low: Mdn = .268 (.51); Mean = .437 (.399)      |

SD = standard deviation; IQR = interquartile range; Mdn = median

With the covariates entered, the effect of group was not significant ( $F(1, 75) = 1.97$ ,  $p = .165$ ) and neither was the effect of condition ( $F(4, 300) = 1.17$ ,  $p = .323$ ). HADS Anxiety was not a significant covariate ( $F(1, 75) = .089$ ,  $p = .766$ ), however, HADS Depression was ( $F(1, 75) = 4.87$ ,  $p = .03$ ). Parameter estimates (beta values) indicated that higher HADS Depression scores were associated with a reduction in the percentage of positive responses.

There were also no significant differences in the proportion of each group showing at least one positive SCR response for neutral ( $\chi^2(1, 79) = 2.79$ ,  $p = .095$ ), negative high arousal ( $\chi^2(1, 79) = 1.69$ ,  $p = .193$ ), negative low arousal ( $\chi^2(1, 79) = .002$ ,  $p = .966$ ), positive high arousal ( $\chi^2(1, 79) = .078$ ,  $p = .780$ ), and positive low arousal ( $\chi^2(1, 79) = 2.22$ ,  $p = .137$ ) images.

### **Amplitude**

Scores for participants who did not show at least one positive response for all conditions were removed from the analysis. The participants included can, therefore, be termed 'responders'. There was no significant difference in the proportion of responders in each group ( $\chi^2(1, 79) = 1.01$ ,  $p = .314$ ). Moreover, removal of two outliers from the control group significantly influenced the results obtained. Therefore, these participants were removed from the analysis. The sample included in the analysis, therefore, consisted of 23 control participants and 20 patients with DS.

None of the potential covariates significantly correlated with SCR amplitude scores for any condition. However, due to the finding that HADS Depression scores had significantly covaried with the proportion of positive SCRs observed in the previous analysis, this variable was entered as a covariate in order to ensure that this possible influence on SCR amplitude was accounted for. The analysis revealed a significant main effect of group ( $F(1, 40) = 5.86$ ,  $p = .02$ ), and a significant main effect of condition ( $F(3.29, 131.6) = 2.81$ ,  $p = .037$ ), on SCR amplitudes. HADS Depression scores were also a significant covariate ( $F(1, 40) = 4.19$ ,  $p = .047$ ), with higher depression scores

being associated with reduced SCR amplitude values. However, the interaction between group and condition was not significant ( $F(3.29, 131.6) = 2.28, p = .077$ ).

The estimated marginal means indicated that the main effect of group was due to significantly higher SCR amplitudes in the DS group (estimated marginal mean = .789, standard error = .105, 95% CI = .577, 1), relative to the control group (estimated marginal mean = .421, standard error = .097, 95% CI = .226, .617). The main effect of condition reflected significantly higher SCR amplitudes for the negative high arousal condition relative to the neutral ( $p = .023$ ), negative low arousal ( $p = .018$ ), and positive low arousal ( $p = .038$ ) conditions. Negative high arousal and positive high arousal conditions did not elicit significantly different SCR amplitudes ( $p = .517$ ). Negative beta values for the covariate across all conditions indicated that HADS Depression scores were associated with reduced SCR amplitudes, as might be expected.

In order to control for the possible effects of medication in the DS group, AED and antidepressant use were added as additional factors in two further runs of the ANCOVA. When AED use was added, the group effect remained significant ( $F(1, 39) = 6.24, p = .017$ ), but the effect of AED use was not ( $F(1, 39) = .537, p = .468$ ). The effect of HADS Depression as covariate was a non-significant trend with AED use in the model ( $F(1, 39) = 3.95, p = .054$ ). When antidepressant use was entered, the effect of group remained significant ( $F(1, 39) = 6.61, p = .014$ ); however, the effects of antidepressant use ( $F(1, 39) = .844, p = .364$ ) and the covariate of HADS Depression ( $F(1, 39) = 1.79, p = .188$ ) were not significant.

### **8.3.5. Exploratory analyses**

The exploratory analyses were carried out only in relation to the SCR amplitude data, because this was the only variable found to differ significantly between groups.

#### ***Correlations between SCR amplitudes and psychosocial / seizure-related variables***

Statistics for the correlations meeting the required alpha level ( $p < .01$ ) are presented in Table 39. As in Chapter 7, partial correlations were carried out to examine

whether the significant correlations remained so after controlling for YoE. In addition, because HADS Depression scores had significantly covaried with SCR amplitude in the present study, this variable was also controlled for in the partial correlations.

**Table 39. Significant correlations ( $p < .01$ ) for SCR amplitude scores (affective picture task)**

|                                               | <b>DS</b> |              |                | <b>Control</b> |              |                |
|-----------------------------------------------|-----------|--------------|----------------|----------------|--------------|----------------|
|                                               | <b>n</b>  | <b>r/rho</b> | <b>p-value</b> | <b>n</b>       | <b>r/rho</b> | <b>p-value</b> |
| <b>Neutral</b>                                |           |              |                |                |              |                |
| HADS Anxiety                                  | 20        | -.614        | .004           | 23             | -.077        | .725           |
| <b>Negative high arousal</b>                  |           |              |                |                |              |                |
| Ictal symptoms – AA*<br>(most severe seizure) | 18        | .611         | .007           |                |              |                |
| MDI ECON                                      | 19        | -.625        | .004           | 23             | .15          | .495           |
| <b>Positive high arousal</b>                  |           |              |                |                |              |                |
| TEC Mean Impact                               | 19        | -.635        | .004           | 23             | -.096        | .662           |
| <b>Positive low arousal</b>                   |           |              |                |                |              |                |
| IASC AD*                                      | 18        | -.751        | .000           | 23             | -.139        | .527           |

HADS = Hospital Anxiety & Depression Scale; AA = autonomic arousal; MDI ECON = Multiscale Dissociation Inventory – Emotional Constriction subscale; TEC = Traumatic Experiences Checklist; IASC AD = Inventory of Altered Self-Capacities – Affect Dysregulation subscale

\* denotes correlations remaining significant after controlling for YoE and HADS Depression scores ( $p < .01$ )

In the DS group, SCR amplitude values for the negative high arousal condition were positively correlated with ictal autonomic arousal symptoms (most severe seizure); whereas they were negatively correlated with emotional constriction scores (MDI ECON subscale). Whilst the former association remained significant after controlling for YoE and depression ( $r = .633$ ,  $p = .009$ ), the latter did not ( $r = -.467$ ,  $p = .068$ ). SCR amplitude for the neutral condition was negatively correlated with anxiety scores (HADS); however, this also did not remain significant in the partial correlations ( $r = -.213$ ,  $p = .397$ ).

For the positive high arousal condition, SCR amplitude negatively correlated with TEC mean impact scores; however, this did not survive correction for YoE and depression scores ( $r = -.302$ ,  $p = .239$ ). Finally, SCR amplitude for positive low arousal images was negatively correlated with affect dysregulation (IASC), and this remained significant after the effects of YoE and depression were taken into account ( $r = -.707$ ,  $p = .002$ ).

### ***Differences between autonomic responders and non-responders***

Exploratory analyses (t-tests, Mann-Whitney or chi-square tests) did not reveal any significant differences ( $p < .01$ ) in demographic factors, psychosocial variables or clinical characteristics (DS group) between ‘responders’ and ‘non-responders’ in either group. Importantly, the proportion of individuals using AEDs or antidepressants did not significantly differ between responders and non-responders.

## **8.4. Discussion**

### **8.4.1. Summary and interpretation**

This experiment was conducted with the aim of further exploring differences in affective processing in patients with DS. A key aim was to investigate affective processing beyond that involving emotional facial expressions. Specifically, the study sought to identify whether there were any differences in affective responses to consciously processed images, displaying varied affectively significant content. The experiment included five conditions, which varied by valence (positive, negative, neutral) and arousal level (high-low). The inclusion of both subjective (valence/arousal ratings) and autonomic (SCR) dependent measures was to permit an examination of subjective reactions concurrently with a more automatic and implicit measure of emotional responsivity. Moreover, a final aim was to carry out exploratory analyses to examine the extent to which any differences on the dependent measures were related to psychosocial factors (e.g. trauma, dissociation) or seizure-related variables (e.g. ictal symptoms, DS frequency).

### ***Subjective responses***

After controlling for relevant covariates, there were no between-groups differences or interactions between group and condition, for either valence or arousal ratings. Furthermore, there were significant main effects of condition for both types of rating. The negative conditions elicited the most negative ratings and the positive conditions elicited the most positive responses, suggesting that the manipulation was successful. Moreover, the negative high arousal condition was rated as most emotionally arousing, whereas the positive low arousal condition was rated as least emotionally arousing. These findings are in accordance with the normative data (see Lang et al., 2005).

Similarly to Roberts et al. (2012), the current findings relating to valence indicated that patients with DS are similar to healthy control participants in the valence (i.e. positivity/negativity) of their subjective responses to affective images. This suggests that a fundamental qualitative difference in conscious/subjective affective responses to significant stimuli was not apparent in the DS group. These findings do not support Hypothesis 1. Furthermore, the lack of between group differences in arousal ratings indicated that the DS group were not different to healthy controls in their conscious experience of emotional arousal in response to the stimuli. This finding does not support Hypothesis 2 and is contrary to the findings reported by Roberts et al. (2012), which suggested that patients with DS may perceive greater intensity in their emotional reactions to positive and neutral IAPS images (particularly compared to controls low in post-traumatic symptoms; PTS). Nevertheless, the findings of the current study are in accordance with a recent report that patients with DS score similarly to healthy controls in the subjective intensity of their emotional reactions, as measured by the Affect Intensity Measure (Urbanek et al., 2014).

However, in Roberts et al's (2012) study, it is possible that the findings could have been related to general psychological distress and psychological symptoms such as anxiety and depression in the DS group, as these were found to be higher in the patients, relative to the low-PTS control group. The investigators did not analyse the extent to which such symptoms correlated with, or predicted intensity ratings, nor were they controlled for statistically (i.e. using ANCOVA). Nonetheless, the DS

group in that study were comparable to the high-PTS control group on most measures of psychological symptoms and yet, differences remained for intensity ratings in response to neutral images.

It is possible that the differences in findings between this study and that of Roberts et al. could be due to the particular images included in the experiments. Images that might be highly emotionally distressing for patients were excluded from the current study (e.g. scenes depicting interpersonal violence) for ethical reasons. Examination of the specific items included by Roberts et al. (2012) indicates very little comparability with the images presented in the present study. Therefore, it is highly likely that this may have contributed to the divergent results. The IAPS stimuli are so varied in content that, unless studies explicitly aim to include the same images, different samples may find the selection of presented images more or less relevant to their specific concerns/life experiences and so, differing affective reactions may be elicited. Moreover, a further difference between the studies was the way in which the stimuli were grouped. Roberts and colleagues grouped stimuli into three conditions (positive, negative, neutral), whereas the images included in the current study were categorised by both valence and arousal. Therefore, it is possible that this difference may have contributed to the differences in findings for arousal ratings.

Another possibility is the influence of trauma. Almost all of the sample included by Roberts et al. (2012) reported a history of trauma and significant PTS, whereas, the current sample was somewhat more diverse with respect to this variable (although a history of trauma was still common). Another difference between this study and that of Roberts and colleagues (2012) was the criterion of video-EEG evidence for diagnosis of DS in the latter, in contrast to a slightly less conservative approach in the current study. It could be the case that the patients in the former study consisted of a biased sample in this regard, possibly comprising more severe or chronic cases, or those for whom diagnosis was more complex on the basis of clinical features.

An additional possible source of difference was the use of a 5-point Likert scale in the study of Roberts et al., and a 9-point Likert scale in the current study. Perhaps, including a larger scale for responses in the current study encouraged participants to

make more fine-grained judgements of their emotional arousal, with more subtle variations than a 5-point scale would allow. Another difference was in the experimental procedure, in that the ITI was set to 15 seconds in the current study, but it was unspecified and reported to average seven seconds by Roberts et al. A longer ITI was included in the current study to ensure that SCR responses had returned to baseline levels before the consecutive stimuli were presented. With a shorter ITI, it is possible that affective arousal in response to individual images had not returned to baseline before presentation of subsequent stimuli. Therefore, it is possible that arousal accumulated in participants undergoing the procedure in the study of Roberts et al. Indeed, it has been found that physiological responses to IAPS stimuli may accumulate with prolonged exposure (i.e. J.C. Smith, Bradley, & Lang, 2005). On the other hand, having a relatively long wait between stimuli in the current study may have allowed participants to experience less ongoing arousal, due to having a period of relaxation between successive stimuli.

To summarise, the currently available findings on subjective responses to consciously perceived affective images (IAPS) in patients with DS are mixed. Both Roberts et al. and the current study failed to find evidence for alterations in the valence (positive-negative) of emotional responses to affective images. However, the findings regarding arousal are more complex, with one study finding greater subjective arousal in response to positive and neutral images in patients with DS (Roberts et al., 2012), in contrast to the current study which found no differences in the subjective experience of arousal to similar stimuli.

The evidence from both studies suggests that patients with DS do not differ from controls in their conscious experience of emotional arousal or valence in response to negative affective images. This latter conclusion is in contrast with Hypotheses 1 and 2 of the current study, which proposed that such differences would be specific to stimuli of this type. Patients with DS also appear to experience similar levels of pleasantness as do controls, in their responses to positive pictures. However, with regards to subjective emotional arousal/intensity, further studies are needed.



### ***Autonomic responses***

Significantly more trials were retained for analysis in the DS group, relative to the control group. However, there were no between-groups effects on the proportion of participants showing positive SCRs, or on the frequency of positive SCRs (i.e. the proportion of trials on which positive responses were observed). Instead, depression was found to covary significantly with the frequency of responses, with higher depression scores associated with lower positive SCR frequency.

On examination of SCR amplitudes exhibited by participants who can be classed as 'responders', a significant group effect was observed. Responders in the DS group had significantly higher mean SCR amplitudes than responders in the control group. This effect was independent of the influence of depression, which was a significant covariate (associated with reduced SCRs). There was a main effect of condition, with SCR amplitude values highest for the negative high arousal condition. However, there was no interaction between group and condition. Furthermore, when relevant medications were added to the model (AEDs, antidepressants), the group effect remained significant. Therefore, it can be concluded that the effects of these medications did not account for the between-group findings. Furthermore, the exploratory analyses indicated that there were no significant differences between responders and non-responders (by group) that might account for these results, including demographics, psychosocial variables, seizure-related and clinical variables.

Therefore, it is apparent that at least a subgroup of DS patients show elevated autonomic reactivity to affectively significant images, and that this tendency cannot be explained by depression or by the most commonly used medications in that group. This finding provides initial support for the hypothesis that elevated autonomic arousal in response to emotionally significant stimuli may be a predispositional, perpetuating and/or triggering factor for DS (see Chapter 3, section 3.6). The observed autonomic hyper-reactivity could contribute to general emotional distress, difficulties in interpersonal relationships and/or coping with stressful life events. Previous research, for example, has indicated that elevated SCRs for threatening IAPS images are associated with the perceived impact of life events in healthy individuals (Najström & Jansson, 2007). The presently reported autonomic hyper-reactivity may

also be linked to previous reports of reduced heart rate variability and elevated HPA-axis activity (see Chapters 2 and 3), for example.

As hypothesised in Chapter 3 (section 3.6), it is possible that an accumulation of autonomic arousal could reach a threshold, at which a DS is triggered. Ongoing autonomic hyper-reactivity to affective stimuli might contribute to such an accumulation. Indeed, the positive association between SCR amplitudes for negative high arousal images and ictal autonomic arousal symptoms, further supports the idea that general autonomic responsivity during highly negative emotional states is indeed associated with the occurrence of such symptoms during DS. However, it should be noted that the current findings appear to apply to a subgroup of patients only. Moreover, of course, the findings from the exploratory analyses must be interpreted with caution due to the possibility of Type I error associated with multiple correlations being carried out on the same dataset. Further research is necessary to examine these relationships in more detail.

The negative correlation observed between SCR amplitudes and affect dysregulation in the DS group is rather difficult to interpret, as a positive correlation between these two variables would be more intuitively plausible (i.e. higher affective dysregulation scores being associated with elevated SCR amplitudes). It is possible that this relationship is mediated by a third variable, most likely depressive symptoms. Nevertheless, the direction of the relationship suggests that elevated affect dysregulation does not seem to contribute to the general elevation in SCRs observed in the main analysis and, therefore, is not discussed further.

#### **8.4.2. Strengths and limitations**

The strengths and limitations of the current study that also apply to one or more of the other studies presented in the thesis are discussed in Chapter 10 (section 10.2). However, there are a number of strengths that are particular to this experiment. This study is one of only two studies that have examined emotional responding to general affective stimuli in this patient group, using laboratory-based experimental techniques. This has extended previous research in the field, which had previously focused on emotional facial expressions only. Moreover, an additional strength was

the use of five separate conditions, in which not only valence, but arousal level were manipulated. This adds to the study carried out by Roberts and colleagues (2012), in which the stimuli were only classified by valence.

As with the experiment described in Chapter 7, an additional strength is the inclusion of both subjective and autonomic (somatic) measures of emotional responding, thereby allowing simultaneous assessment of these response domains in the same participants. Another strength of this study was the fact that relevant neuropsychological tests were administered, in order to ensure that any differences observed could not be attributed to cognitive factors. Moreover, the use of statistical control for relevant psychological variables (i.e. depression, anxiety) allowed a more rigorous test of the hypothesis that differences were associated with a diagnosis of DS, over and above the presence of general psychological distress in the DS group.

On an ethical level, this study aimed to ensure the emotional wellbeing of participants by excluding images that might have caused acute emotional distress. Indeed, all participants reported that they had not felt unusual levels of distress either during or after completion of the experiment. Therefore, it can be concluded that the emotional wellbeing of the participants was maintained by taking a relatively conservative approach to stimulus selection. However, this could also be seen as a limitation. Patients with DS may differ from controls specifically in their responses to particular types of stimuli, such as those of relevance to their trauma histories. As such, excluding particular groups of stimuli may limit the potential for observing important differences between patients with DS and control groups. Moreover, it could be argued that stimuli should be selected to be specifically relevant to the patient group, in a personalised fashion, as is the case in some studies of PTSD and BPD that involve personalised trauma scripts (e.g. Elzinga et al., 2003; Schmahl et al., 2004). Perhaps, in order to fully understand DS, and how aetiological factors relate to emotional responsivity in this group, it may be necessary to include stimuli that are likely to elicit stronger responses.

The study had a few other limitations. One possible weakness could be a loss of power linked to the inclusion of five different emotional conditions. Whilst this

allowed a more detailed analysis of the possible effects of valence and arousal level, perhaps categorising stimuli on just one of these dimensions (as in Roberts et al., 2012) may have allowed the retention of greater statistical power. It should be noted that the exploratory correlational analyses with SCR amplitudes may have been statistically underpowered (see Chapter 4, section 4.5.2), as was the case in Chapter 7. It might also be argued that the inclusion of only six images in each condition was insufficient. Initially, eight images had been included; however, the number was reduced after the pilot study due to the experiment being too lengthy, with participants losing concentration. The length of the experiment was also increased by necessity of a long ITI due to the measurement of SCRs. A future possibility could be to conduct a study that involves subjective responses only, which would allow a greater number of trials in each condition and shorter ITIs. Limitations relating to the psychophysiological measurements were discussed in Chapter 7 (section 7.4.2).

#### **8.4.3. Conclusions**

The findings of this study suggest that patients with DS are similar to healthy controls in their subjective emotional reactions to general affective images. However, those participants who were deemed to be autonomic ‘responders’ in the DS group displayed significantly elevated SCR amplitudes relative to ‘responders’ in the control group. The use of AEDs or antidepressants, and depressive symptoms did not account for this finding. Therefore, the study shows that at least a subgroup of patients with DS exhibit exaggerated autonomic responses to general affective stimuli, possibly on an ongoing basis. Combined with the subjective findings, this suggests that elevated somatic emotional responses occur in the absence of altered subjective emotional responses, in this subgroup. This pattern might reflect a dissociative mechanism, in which patients’ conscious awareness of their somatic responses to affective stimuli is disrupted. Hypothetically, this could allow autonomic arousal to build up over time, without patients being aware of it. As suggested in Chapter 3 (section 3.6), such an accumulation of somatic arousal might contribute to triggering DS on an ongoing basis. The correlation between elevated SCR amplitudes for negative high arousal images and ictal autonomic arousal symptoms, tentatively supports this hypothesis. However, further examination of this relationship warrants further research.

## **Chapter 9. Emotion and dissociative seizures: a phenomenological analysis of patients' perspectives**

### **9.1. Introduction**

The quantitative findings presented in previous chapters suggest a complex and potentially divergent style of emotional processing in patients with DS. Whilst the use of quantitative measures has several known strengths (i.e. large data sets, efficient data collection, reduction of bias in data collection/analysis, hypothesis testing), an important limitation in the exclusive use of such measures is the lack of insight gained into the phenomenological experience of the individuals being studied. In other words, the person behind the numbers is obscured, and the many valuable insights they might provide are not given adequate examination. Qualitative techniques can be used to provide an insight into the unique meanings that individuals ascribe to their experiences (Willig, 2013).

Increasing numbers of investigators have adopted qualitative techniques in studies of DS in the last decade, often based on in-depth interviews with patients, or qualitative analysis of patients' interactions with clinicians. For example, several investigators have used such techniques to examine patients' reactions to receiving the diagnosis of DS (Carton et al., 2003; Karterud et al., 2010; R. Thompson et al., 2009; Wyatt et al., 2014). These studies highlighted several themes in patients' responses to the diagnosis, particularly feelings of confusion about the nature of the disorder, but also those of relief and feeling like a 'normal' person again (i.e. due to not having a chronic neurological condition). These findings have important implications for the ways in which diagnoses are communicated, and the extent to which patients are supported thereafter. Issues relating to provision, and patients' experiences of treatment, have also been examined qualitatively (e.g. Baxter et al., 2012; Fairclough et al., 2014; Quinn et al., 2010; McMillan et al., 2014; Wyatt et al., 2014).

Other authors have adopted qualitative techniques in investigating DS patients' experiences and understanding of their disorder. The study by Carton et al. (2003), for example, reported themes relating to patients' conceptualisations of their attacks.

Some patients experienced their seizures as a release of accumulated emotion, stress, or as in some way related to previous traumatic experiences. However, patients were also often confused about the way in which these processes inter-related, particularly as they often noted a lack of temporal contiguity between the triggering event and the occurrence of the DS. Moreover, patients reported a range of factors that seemed to contribute to the aetiology of the disorder, such as stressful events (e.g. pain, interpersonal problems), the experience of anxiety or panic symptoms, and a possible disproportionate allocation of attention to bodily processes. Many of the sample also reported considerable negative consequences of DS on other areas of life, such as employment, self-esteem, social isolation and anxiety.

Dickinson and colleagues (2011) used thematic content analysis to explore patients' perspectives on their disorder and noted that a theme emerged whereby patients linked their disorder to both early (e.g. head injury, physical assault, exposure to epilepsy) and recent (e.g. divorce, bereavement, legal proceedings) life events. Again, many patients expressed confusion about the aetiology of their disorder. Moreover, Green et al. (2004) also noted considerable confusion in patients' accounts of their disorder, pertaining to both terminology and the possible causal processes involved. These latter authors also noted a distinct tendency to discuss DS dualistically as either organic or psychological, rather than acknowledging the possibility that psychological factors can interact with physical processes. Two studies also sought to examine patients' representations of their illness by examining the ways in which such patients described their attacks during interactions with clinicians (Plug et al., 2009; Robson et al., 2012), with important differences observed in the ways in which patients with ES and DS talked about their attacks.

It is clear that qualitative techniques can yield a range of valuable insights, many of which having important clinical implications. Furthermore, qualitative studies can generate hypotheses for testing with quantitative techniques. In addition, such studies provide an important opportunity for the involvement of patients in the research process, ensuring that their views are represented in a formal medium and thus given validation. During the process of conducting the quantitative aspects of the research presented in this thesis, many patients were keen to share their perspectives on how

their emotional states might, or might not, relate to the onset of their disorder and the ongoing occurrence of seizures. The aim of the present study was, therefore, to present idiographic data as a means of enriching the findings presented in the thesis. The study sought to explore the following research questions:

1. How do patients with DS perceive their general emotional functioning?
2. To what extent can patients with DS reflect on and understand the possible role of emotions in the onset of the disorder and/or ongoing seizure generation?
3. How do patients with DS perceive their ability to recognise and understand the emotions of others?

In order to investigate these questions, semi-structured interviews were conducted with a subsample of patients who had completed the quantitative aspects of the research.

## **9.2. Methodology**

The research followed as closely as possible the recommendations made by proponents of Interpretative Phenomenological Analysis (Smith & Osborn, 2008). The details below can be assumed to be in accord with such recommendations, unless otherwise stated.

### **9.2.1. Participants**

Recruitment for the study commenced in January 2011 and ended in August 2012. Eligibility criteria were as outlined in Chapter 4 (section 4.2) for the DS patients. All patients who had completed the quantitative aspects of the research during the period outlined above were offered an information sheet for the present study in person and, if interested, were contacted within three weeks (but no less than one week) after completing the quantitative session.

As suggested by Smith and Osborn (2008), there is no single correct sample size for studies using IPA. The sample size ( $n = 15$ ) included in the current study was within the range that has been used in other such studies, with recruitment terminating when no additional themes emerged during preliminary analyses of the transcripts.

### **9.2.2. Procedure**

All patients provided written informed consent on arrival at the interview location (see Appendices 7 and 8 for the information sheet and consent forms respectively). Data were collected with semi-structured interviews lasting between approximately 30 and 90 minutes. Fourteen of the interviews took place in a psychology laboratory, and the remaining interview was conducted in the patient's home. An interview schedule was designed prior to data collection, in order to ensure that the interview covered topics of relevance to the research questions outlined above. The interview schedule can be found in Appendix 20. The schedule was used as a guide only, being applied flexibly depending on the particular topics on which each participant wished to elaborate. The schedule included open-ended questions of direct relevance to the research topic, in addition to potential prompts for use only if necessary. The same schedule was utilised in all interviews.

The interviews were digitally recorded, with patients' permission. On completion of the interview, participants were given the opportunity to ask questions. Patients' wellbeing was checked before leaving the building. The same interviewer (the research student) carried out all interviews. The interviewer was a British Caucasian female postgraduate student (SP), in her late twenties/early thirties during data collection.

### **9.2.3. Data analysis**

All interviews were transcribed verbatim by the research student, as soon as possible after they were completed. Transcriptions were anonymised by giving each participant a unique participant number. The transcripts included all spoken words, pauses, and non-verbal utterances (e.g. laughs), but not prosodic features (as recommended by Smith & Osborn, 2008). In IPA, the analyst engages actively with the transcript and aims to interpret the meanings implicit in the data. The following



steps are recommended in IPA and are, therefore, the steps that were taken in analysing the data reported here.

### **1. Identification of themes in the first transcript:**

The first transcript was read several times, with initial notes being made on the transcript freely when sections were thought relevant or meaningful. No 'rules' governed how the initial notes were made, with annotations including paraphrasing, possible meanings, repetitions, associations, contradictions, use of language, among others. After this, the transcript was read again, and emergent themes noted, with specific phrases or words being selected to encapsulate categories of notes made in the first stage.

### **2. Theme connection:**

A list of possible themes was devised, based on those emerging from the first transcript, but listed separately (in a new document). The list of possible themes was examined for potential inter-relationships, with some of the themes seeming to cluster together, whereas others were relatively unique. As themes were organised into categories and clusters, these were checked back against the transcripts to ensure that the meaning of the emerging theme classification accurately reflected what the participant had said. During this process, relevant quotations from the text were placed under relevant theme titles. The clusters of themes were categorised into superordinate themes at this point.

### **3. Adding additional transcripts to the analysis**

The themes developed from the first transcript were used as an initial classification by which to examine the second transcript. Similarities were sought and relevant quotations from the second transcript were added to the relevant existing theme. In addition, new emerging themes from the second transcript were added to the overall classification, where no overlap was present with existing themes. This process continued with all subsequent transcripts, until no new themes seemed to emerge. A final classification scheme of themes was then developed, checking this back against the raw data from the transcripts. The classification was refined by including those superordinate themes and subthemes that best reflected the transcripts included.

#### **9.2.4. Quality and reflexivity**

During data analysis and writing up of the results, notes were made when there was a possible influence of the research student's background or characteristics on interpretation of the data; efforts were made to minimise the influence of such characteristics on the interpretations made. The research student's primary supervisor (LG) independently examined a subsample of transcripts, blind to the themes that had been identified by the student. The themes identified by the supervisor were very similar to those identified by the student; however, some small modifications were subsequently made to the hierarchy of themes. Furthermore, a preliminary summary of the main findings was sent to two patients who had participated in the study (with prior consent agreed). One of the patients responded, confirming that the summary was understandable, acceptable and reflected her viewpoints on the topics discussed.

### **9.3. Results**

#### **9.3.1. Demographics**

Fifteen participants were included in the study. The demographic characteristics and duration of DS disorder for each participant are presented in Table 40.

#### **9.3.2. Overview of themes**

Several superordinate themes emerged during the analysis, partially reflecting the types of question included in the interview schedule. These were themes relating to: patients' general emotional functioning, the relationship between emotions and DS, stressful/adverse life events, relating to others, resilience, protective factors, and coping strategies. The themes and subthemes are presented with representative quotations in Tables 41 to 45. Some of the sub-themes seemed to fit into more than one category; however, for simplicity, they were placed under the superordinate category that best represented the meaning of the theme for the patients. Where this is the case, the cross-over is discussed in the text.

### **9.3.3. General emotional functioning**

One cluster of themes related to patients' perceptions of their general emotional functioning (see Table 41). These included the frequent experience of negative affect, affect dysregulation, inhibited emotional experience/expression, rumination, and a strong connection between emotion and bodily states. For some, the phenomena described seemed to represent long-term tendencies rooted in development. However, other patients indicated these experiences were more specific to a given time in their lives. The difficulties in emotional functioning were directly attributed to the ongoing experience of seizures in some cases. Patients often felt that the experience of the chronic and severe seizure symptoms had caused a deterioration in their emotional well-being. In some instances, the degree of deterioration in emotional state since the onset of DS led to patients' perceiving alterations in personality and sense of 'self' as a consequence, described as being like 'a different person'.

#### ***Negative affect***

A theme common to almost all patients was the experience of aversive affective states. Patients often felt that their current experience of negative affect was exacerbated by, or a direct consequence of the symptoms of DS or other somatic concerns (statements 1 and 2). Moreover, others described previous periods of depression in relation to stressful/traumatic life events or circumstances (statements 3 and 4). Some patients described a specific lack of emotional wellbeing and positive affect, or 'happiness', that appeared to be relatively long-standing (statements 5 and 6).

#### ***Affect dysregulation***

Lability of mood state was also a common feature, with patients describing frequent extremes of emotion often occurring within short periods of time (i.e. a day), labelled 'ups and downs' (statements 7 – 10). One patient felt that this tendency had worsened since the onset of DS (statement 11).

**Table 40. Participant characteristics (qualitative study)**

| <b>Participant number</b> | <b>Age</b> | <b>Gender (M/F)</b> | <b>Ethnic background</b> | <b>Occupational history</b> | <b>Educational background</b> | <b>Time since DS onset (months)</b> |
|---------------------------|------------|---------------------|--------------------------|-----------------------------|-------------------------------|-------------------------------------|
| <b>1</b>                  | 40s        | F                   | White British            | Self-employed/homemaker     | GCSEs / O levels              | 48                                  |
| <b>2</b>                  | 40s        | F                   | White British            | Homemaker/professional      | Undergraduate degree          | 84                                  |
| <b>3</b>                  | 50s        | F                   | White British            | Homemaker                   | None                          | 120                                 |
| <b>4</b>                  | 30s        | M                   | White British            | Professional                | Postgraduate degree           | 33                                  |
| <b>5</b>                  | 30s        | F                   | White British            | Administration              | Undergraduate degree          | 192                                 |
| <b>6</b>                  | 50s        | F                   | Mixed                    | Professional                | Postgraduate degree           | 84                                  |
| <b>7</b>                  | 20s        | F                   | Black African            | Professional                | Postgraduate degree           | 84                                  |
| <b>8</b>                  | 30s        | F                   | White British            | Homemaker                   | GCSEs / O levels              | 120                                 |
| <b>9</b>                  | 50s        | F                   | White British            | Professional                | Undergraduate degree          | 44                                  |
| <b>10</b>                 | 30s        | M                   | White British            | Intermediate occupation     | GCSEs                         | 15                                  |
| <b>11</b>                 | 50s        | M                   | White British            | Unemployed/disability       | None                          | 276                                 |
| <b>12</b>                 | 30s        | M                   | Mixed                    | Intermediate occupation     | Diploma                       | 168                                 |
| <b>13</b>                 | 30s        | M                   | White British            | Service occupation          | None                          | 192                                 |
| <b>14</b>                 | 20s        | F                   | White British            | Unemployed/disability       | None                          | 120                                 |
| <b>15</b>                 | 50s        | F                   | White British            | Intermediate occupation     | GCSEs / O levels              | 30                                  |

M = male; F = female

**Table 41. Themes relating to general emotional functioning**

| <b>Theme</b>         | <b>Sub-theme</b>                                                     | <b>Typical statements</b>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            |
|----------------------|----------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Negative affect      | Current negative affect related to somatic symptoms (including DS)   | 1. <i>"Sometimes I allow myself to have bad days....I just feel horrific and awful, and I just wallow..."</i> (P1)<br>2. <i>"I get...especially since all my health issues started, I get very depressed, due to all the things that are going on with me."</i> (P12)                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                |
|                      | Previous depression related to difficult life events / circumstances | 3. <i>"I recognise I was depressed as well, and I didn't have any sort of help from that..."</i> (P9)<br>4. <i>"I used to be in huge emotional pain...my life was really hard emotionally...I went through a very difficult time, I was depressed..."</i> (P6)                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       |
|                      | Long-term lack of positive affect                                    | 5. <i>"It's been like that, off and on, it's more negative than it is positive...since I was really young..."</i> (P13)<br>6. <i>"I wouldn't say I've ever felt really happy...I wouldn't say, for the past like 15 or 20 years, I've been happy, I can't remember a time in my life..."</i> (P7)                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    |
| Affect dysregulation | 'Ups and downs'                                                      | 7. <i>"I'm not, I don't think I've ever been sort of somewhere 'in the middle', for a very long time it is up and down...It's just constant, it can be constant, so in one day it can be: you get up in the morning, you'll feel fine, but then something happens or your mood might suddenly change, for no reason, you feel a bit lower, and that's how it is...."</i> (P8)<br>8. <i>"My bad moods are...they're really bad...When I'm in a bad mood, I'm really unhappy. It's severe. When I'm in a good mood...nothing can go wrong."</i> (P14)<br>9. <i>"I could be feeling fine, and somebody could say something to me, and I could go down, depending on, you know, what they're saying."</i> (P2)<br>10. <i>"Just the little things, can make my day like, the worst day ever really."</i> (P7)<br>11. <i>"...before my attacks, I was quite wavy anyway....as opposed to a calm sea...but since the attacks...my emotions have changed, are much more dramatic...the wavy lines are much higher and much lower, at either end of the spectrum..."</i> (P1) |

|                                                        |                                                         |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      |
|--------------------------------------------------------|---------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
|                                                        | Episodic excessive emotional expression                 | <p>12. "I cry, if I get, if it got too much, I'll cry. I sit and cry." (P3)</p> <p>13. "I kind of, blow up at inappropriate things at home. Well not inappropriate, but really small things at home." (P5)</p> <p>14. "...if someone brings up the past that's mainly when it happens, that's when I'll get aggressive and go at someone." (P14)</p> <p>15. "You know, it comes out of me...in a sense where, I'll smash a plate or a cup." (P11)</p>                                                                                                                                                                                                                                |
|                                                        | Difficulties calming down linked to inhibiting emotions | <p>16. "I'm not good at calming down. It takes me forever...when I hold things in, and think what I'm saying, and I have to hold back a little bit, that's when I can't calm down." (P8)</p> <p>17. "Once I cross the line, then it's avalanche...and I think that's because I bottle it all up...so, there is a line. The point of no return I suppose." (P2)</p>                                                                                                                                                                                                                                                                                                                   |
| Inhibited experience / expression of negative emotions | Shutting off difficult emotions                         | <p>18. "I have to accommodate certain negative influences, so I think they leave me flat, rather than down. Numb. Shutting it off. Like, blocking it." (P2)</p> <p>19. "...even before I had seizures, over the years I couldn't understand that at times of great stress I'd always felt quite well and happy...." (P15)</p> <p>20. "I very much compartmentalise stuff...with very strong emotions, be it very good ones or very bad ones, I tend to close off from them... (P4)</p>                                                                                                                                                                                               |
|                                                        | Bottling-up emotions and putting on a 'brave face'      | <p>21. "I tend to not show what I feel...when I'm not happy I don't want people to know that I'm not happy." (P7)</p> <p>22. "I don't like trusting my true emotions to people...." (P2)</p> <p>23. "Throughout my entire life and throughout any stresses and strains I've had, the way I've always dealt with it, is – the worse I'm actually feeling, the happier I will be to people around me. I hide my feelings very much so...I just, you know, put on this sort of happy face..." (P15)</p> <p>24. "But even my best friends, you know, they've come to see me, so I don't have to be completely, you know 'painted smile', but there's still an element of that." (P1)</p> |

|                                              |                                                           |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               |
|----------------------------------------------|-----------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Inhibition of emotions rooted in development |                                                           | <p>25. <i>"I think, one of the things about my family life, and my upbringing, was that very strong emotions are not ok, and they're not shown. Which I guess ties into the seizures in some way."</i> (P4)</p> <p>26. <i>"I feel myself, it's about how I was emotionally as a child...because I was very sensitive and the problems that I faced, I suppressed that. I forget about that for many many years. You know, it went out of my mind completely."</i> (P6)</p> <p>27. <i>"Again, I don't really like to show my emotions in front of him...as a kid he always told me 'don't cry in front of people, girls don't cry'...He is a very shut-down person..."</i> (P5)</p> <p>28. <i>"Erm, my own personal background, even at that time, meant that I had no control over my situation...so I didn't talk about it all the time. In fact, I think I was so successful at getting other people to talk to me, because it stopped them talking about me."</i> (P2)</p> |
|                                              | Insights into links with seizures / negative consequences | <p>29. <i>"Sometimes...I want to hold things back...but I know, because of the seizures and everything, I know that's the worst thing you can do, to keep it all in to yourself. You've got to speak to someone about it."</i> (P8)</p> <p>30. <i>"So it was all about carrying on, covering up, not being able to say anything. Particularly in some very stressful situations...I know that was internalising the emotions, and...I know how it affected me too."</i> (P2)</p>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              |
| Rumination / intrusive thoughts              |                                                           | <p>31. <i>"...it just starts getting worse, I just start thinking about it, and the negative thoughts start getting even worse and worse and worse."</i> (P13)</p> <p>32. <i>"I have to keep processing it. I keep processing it, over and over and over...it's like a little nag, and it won't go away, no matter what I try to do, it won't go away. It's like I have to keep thinking about it..."</i> (P9)</p> <p>33. <i>"I analyse things a lot...I tend to attribute reasons...."</i> (P2)</p>                                                                                                                                                                                                                                                                                                                                                                                                                                                                          |
| Emotions and physical symptoms (non-DS)      | Pain                                                      | <p>34. <i>"I'm always getting the pain...it's just like all of the time, even for small things, if I get angry or something like that....it's just like very quick, and then I feel very tense....it can take days before the pain goes...."</i> (P7)</p>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     |

|                                                    |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              |
|----------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Motor symptoms                                     | 35. <i>"I get these jerks, you know, and I recognise if I'm feeling a bit anxious, it's like a build-up, and my body will jerk, and then the anxiety goes."</i> (P9)                                                                                                                                                                                                                                                                                                                                         |
| Exhaustion                                         | 36. <i>"...if something's upset me...perhaps the day after, or the day after that, I will feel very exhausted...and you know, if I allow myself time enough, then it will make me feel physically not well..."</i> (P15)                                                                                                                                                                                                                                                                                     |
| Sleep disturbance                                  | 37. <i>"If I'm upset I won't sleep...and then when I do sleep I have really strange weird dreams that are sort of connected to what I'm upset about..."</i> (P5)<br>38. <i>"I mean night-times, there's the night traumas, the nightmares, night sweats...there is a connection with the nightmares with the bullying."</i> (P13)                                                                                                                                                                            |
| Physiological responses to acute affective arousal | 39. <i>"If I'm particularly angry or excited...I can feel a change in my heart beat..."</i> (P12)<br>40. <i>"...when I get angry, I get a bit shaky...I've had a lot going on this week, to the point the other day, when I was very angry, I was shaking..."</i> (P8)<br>41. <i>"Sometimes I get hot, and I tend to, it's sometimes like the anger is boiling up inside me, and my heart races a bit, if it's something really bad...If I get really angry, I shake, which is quite embarrassing."</i> (P5) |

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P = participant number



Discrete episodes of excessive emotional expression were also a commonality in the sample, including weeping (statement 12), aggression (statements 13 and 14), and destructive behaviours (statement 15). Some patients also recognised that they experienced difficulties in calming down once upset, and that these patterns might be linked to the ongoing ‘bottling-up’ of their emotions (statements 16 and 17). The ‘ups and downs’ and excessive emotional expression described above were attributed to specific triggers by some patients, including reminders of past life events (statement 14), interpersonal interactions or conflict (statement 9), and other daily life events (statements 7, 10, and 13).

### ***Inhibited experience and expression of negative affect***

A very common theme emerged pertaining to a marked pattern of inhibited experience of negative emotion. This was most commonly referred to as ‘closing down’ or ‘shutting off’ unwanted feelings or mood states (statements 18 - 20). This theme was linked to another coping strategy involving behavioural/social ‘shutting down’ (see section 9.3.7).

Patients frequently talked of “bottling-up” unpleasant emotions, that is, inhibiting expression of such emotions to others (statements 21 and 22). A linked feature was that of “putting on a brave face”, presenting an artificially positive, cheerful or confident persona to others, despite this being incongruent with the actual affective state being experienced (statements 23 and 24). This sub-theme was connected to a belief that the expression of feelings and emotions might be a burden to others; therefore, for some patients, inhibited expression of emotion was a means of trying to ‘protect’ others (see section 9.3.6. below).

These tendencies towards experiential or expressive emotional inhibition were often described as long-standing (e.g. statement 23), and were attributed to events/situations that had occurred during childhood/adolescence in some cases (statements 25 – 28). Some patients referred to a possible relationship between these tendencies and the occurrence of seizures (statement 25), and/or indicated that they were aware of the maladaptiveness of these patterns (statements 29 and 30).

### ***Rumination***

Rumination about unpleasant emotions or upsetting experiences were commonly described (statements 31 – 33). For some, these ruminative thoughts were referred to as somewhat intrusive and not within voluntary control (e.g. statement 32). Whereas, for others, this tendency was described as more of a controlled analysis of emotional episodes (e.g. statement 33).

### ***Emotions linked to physical feelings***

Many patients perceived strong interconnections between emotional states and physical symptoms and experiences. For example, emotion was described as causing physical discomfort or pain (statement 34), motor symptoms (statement 35) and/or tiredness/exhaustion (statement 36). Several patients also experienced sleep disturbances such as insomnia and night-terrors as a result of stress or anxiety (statements 37 and 38). Moreover, some patients were able to describe acute awareness of physiological changes (e.g. heat, shaking, heart-racing) during acute emotional arousal (statements 39 – 41).

### **9.3.4. Stressful/adverse life experiences or circumstances**

Patients reported a wide range of stressful and/or adverse events and circumstances. For some, isolated and remote traumatic incidents were discussed, whereas for others there were more generalised or ongoing problems. Several themes emerged in relation to this topic, reflecting the key areas in which patients experienced difficulties. These included interpersonal problems, the experience of abuse, somatic concerns and symptoms, and generally stressful circumstances. Table 42 includes examples of patients' statements.

### ***Relationship/interpersonal problems***

Some patients described current relationship problems, including conflict (statements 42 and 43), intimate relationship breakdown (44) and difficulties in forming relationships with others (statement 45). Other patients referred to relationship problems that had affected them in the past (statements 46. and 47).

**Table 42. Themes relating to stressful/traumatic life experiences or circumstances**

| <b>Themes</b>                         | <b>Sub-theme</b>                                            | <b>Typical statements</b>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     |
|---------------------------------------|-------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Relationship / interpersonal problems | Relationship dysfunction / interpersonal problems (current) | <p>42. <i>"I know for example that there is an ongoing situation with me, where conversations are being misrepresented...and it feels terribly unjust and terribly unfair...and this has gone on for several years." (P2)</i></p> <p>43. <i>"Me and (anon) don't have a good relationship...And we will row from the moment we get up to the moment we go to sleep." (P5)</i></p> <p>44. <i>"My partner said he was moving out, so that's what happened." (P8)</i></p> <p>45. <i>"I find it hard to actually make friendships. I mean I try and get out there, around people, and I just can't do it." (P13)</i></p>                                                                                                                                                                                                                                                                                                                                                                          |
|                                       | Relationship dysfunction (previous)                         | <p>46. <i>"...when my parents got divorced...it's quite a wounding thing...you know, I was very cross and very bitter, and I carried that for a long time." (P1)</i></p> <p>47. <i>"...for the first five or maybe six years of my marriage, I cried constantly because it was so awful...it just destroyed me...." (P15)</i></p>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             |
| Abuse                                 | Childhood abuse                                             | <p>48. <i>"The only thing I can't understand...is when a person hits a child. I really do not tolerate that. You know, I have to say something. Because of having been a battered child myself." (P11)</i></p> <p>49. <i>"I was in an abusive childhood, so my (anon) was an alcoholic, you can't explain that to your friends. So, you bury it..." (P2)</i></p> <p>50. <i>"I think...when I was a child, I went through quite a bit of child abuse...and I think I locked that away, and I know because I just got on with it...my way was to kind of get on with it...I don't think on it at all, sometimes I'm kind of, not angry, I'll be upset that I had to suffer that, and now it's still affecting my adult life." (P10)</i></p> <p>51. <i>"So, when she, when I was beaten, I hate her, I hate the pain, and I hate not being able to express my pain, my physical pain, my emotional pain, I hate not being able to cry, I thought that was, you know, pure cruelty." (P6)</i></p> |

|                                   |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          |
|-----------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Adulthood abuse                   | <p>52. "...he'll shout and he'll scream, and throw things...last week I was making a drink and I'd got the boiling kettle in my hand...and he just walked up behind me and pushed me." (P5)</p> <p>53. "I just went out for that walk, early in the morning, walked around the corner, bang bang bang, got my head kicked in, got beaten up..." (P11)</p> <p>54. "...and then the intimidation started and the mental bullying started...and that was it." (P13)</p> <p>55. "When I think back, there was a (anon) there (workplace) who was a bit of a bully really...and there was an incident when she really shouted at me in front of a lot of people...." (P9)</p> |
| Generally stressful circumstances | <p>56. "...currently my situation at the moment is quite stressful..." (P2)</p> <p>57. "I've had so many things happen, to me, during my life..." (P1)</p>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               |
| Somatic symptoms and concerns     | <p>58. "Oh I've always got aches and pains, because of the arthritis that I have, on a day-to-day basis....there's always elements of pain in my daily life..." (P1)</p> <p>59. "Most days, I end up with a bad headache throughout the day...I've been to the neurologists and I've told them about it, and I've had brain scans, and they're just not finding anything." (P13)</p> <p>60. "I have pain all the time. Like in my leg, it's just like, all the time. Nobody can do anything about that..." (P7)</p>                                                                                                                                                      |
| Sleep disturbance                 | <p>61. "I very rarely sleep anyway. I haven't been asleep at all last night, and I had about an hour and a half the night before." (P12)</p> <p>62. "I don't go more than probably three, three and a half hours a night...the mind's constantly active." (P10)</p> <p>63. "Sleep, that's the main thing at the moment....I keep waking up constantly, for no reason, and sometimes you think it's time to get up...and then I can't get back to sleep again." (P8)</p>                                                                                                                                                                                                  |

P = participant number

### ***Abuse***

A recurrent theme was the experience of interpersonal abuse, often during childhood (statements 48 - 51). Some patients described having 'locked away', buried or suppressed these experiences and appeared to have some insight into how the experiences had affected them (statements 49 and 50). For some patients, abusive experiences had also occurred during adulthood, including domestic abuse (statement 52), physical assault (statement 53), and workplace bullying (statements 54 and 55).

### ***Generally stressful circumstances***

Some patients tended to describe their lives as generally stressful, and/or traumatic. For some, there was a chronic or long-term nature to the stressors and often multiple stressors or traumas were reported. Rather than discussing specific incidents, these patients made more general references to difficult circumstances or events (statements 56 and 57).

### ***Somatic concerns and symptoms***

Almost all of the patients described additional somatic illness, symptoms or concerns. The most commonly reported were pain (statements 58 – 60) and sleep disturbances (statements 61 – 63).

### **9.3.5. The role of emotions in DS**

A number of perspectives were expressed in relation to the ways in which patients felt that their emotions might be linked to the occurrence of DS. These themes included linking DS occurrence to life stressors and traumatic events, viewing DS as a release of built up emotion, and an inconsistent temporal relationship between emotional triggers and DS. Furthermore, several patients described emotions/experiences that commonly occurred post-ictally. Representative quotations can be found in Table 43.

### ***DS onset linked to stress / trauma***

Stressful circumstances or elevated stress levels were often described as having occurred at the time of the onset of the disorder, or leading up to that point (i.e. first seizure occurrence). Such experiences included medical/somatic crises or

procedures (statements 64 – 67), traumatic life events (i.e. physical/sexual assault; statements 68 and 69), and relationship breakdown or crises (statements 72 and 74). Other stressors included moving home (statement 71), occupational stress (statement 73), and bereavement (statements 70 and 73) and physical/mental exhaustion (statements 72 and 73). Some patients described multiple life stressors occurring prior to DS onset (statements 70 - 73).

### ***DS as emotional ‘relief’ after a ‘build up’***

DS were associated with switching off from emotional distress or strong emotions by several patients (statements 75 - 76). Some patients felt that their seizures had originally been triggered by a build-up of emotional distress (statements 77 and 78). Feelings of well-being or relief following a seizure were also described (statements 79 and 80).

### ***Inconsistent emotional triggers for DS***

The relationship between DS occurrence and emotional states or triggers was perceived as unpredictable by the majority of patients, with DS sometimes occurring at times of stress or anxiety and sometimes not (statements 81 and 82). Indeed, in several cases, patients specifically described having seizures during times in which they felt happy or relaxed (e.g. statement 82). One patient mentioned that her DS used to be preceded by fear consistently, but that now they seemed to be triggered by the experience of any strong emotion, rather one consistent emotional state (statement 83).

### ***Post-ictal emotions / experiences***

During DS, the seizures were most often perceived as a state of total lack of emotion, during which they reported not feeling anything at all and ‘shutting off’ from their feelings (see previous section). On the other hand, emotional reactions and experiences after DS were more common, including frustration (statement 84), tiredness (statements 85), weeping (statement 86), but also relief in some cases (see previous section).

**Table 43. Themes relating to the role of emotions in DS**

| Theme                                                | Sub-theme               | Typical statements                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              |
|------------------------------------------------------|-------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| DS onset linked to stressful / traumatic life events | Medical/somatic crises  | 64. "...they followed a long period of intense physical pain. The pain came first, the attacks came after." (P2)<br>65. "I had to go into hospital for minor surgery, I had general anaesthetic, I came out of the anaesthetic, and ended up staying in for a week...they hadn't a clue what it was, and it kind of all went from there..." (P1)<br>66. "I got sick, and I was told I had meningitis...and so, they started around then...." (P5)<br>67. "In 2004 I had a major physical failure. I had the seizures, I had the stomach, I had to have surgery, I had breast cysts, I had cysts on my uterus...." (P6)                                                                                                                                                                          |
|                                                      | Physical/sexual assault | 68. "I'd been attacked, shortly before that..." (P5)<br>69. "I was ok-ish then. I just went out for that walk...got beaten up...it all stemmed from there..." (P11)                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             |
|                                                      | Multiple stressors      | 70. "Prior to all that, I lost my wife, my daughter, er, I gave up the alcohol...yeah so I was sort of, I'd had a few sad moments, bereavements, before the actual...erm, the first seizure." (P11)<br>71. "I had a lot going on...moving home, getting married, and all that sort of thing, so...it was stressful, at that time." (P8)<br>72. "For me, they came at a time when my body was under a huge amount of strain from stress, and also I ran the marathon...my relationship broke down in the March, I ran the (marathon) in the April, and I was scheduled for the surgery in June...physically, there was a lot of stuff going on..."(P4)<br>73. "I was over-loaded...I was working ten hours a day...I was exhausted, exhausted. So, it happened that my grandmother died..." (P6) |
|                                                      | Relationship crises     | 74. "I can remember my first day, my first seizure started...the problem I was facing at home had just got to a point where I just couldn't deal with it any more...I got to my limit...something had to give, and for me, it obviously manifested in a seizure." (P15)                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         |

|                                                                        |                                                         |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     |
|------------------------------------------------------------------------|---------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| DS as emotional 'relief' or 'shut-down'                                | DS related to switching off from emotions               | <p>75. "So it would seem like to have a seizure would take me out of my surrounding problems... Switched off, emotionally switched off, I don't feel anything... I don't feel unhappy or I don't feel happy, I just feel as though somebody has literally switched me off and I'm not worried about anything." (P15)</p> <p>76. "...I'm definitely aware that it's at times when I do shut-off emotionally, that they come on..." (P4)</p>                                                          |
|                                                                        | DS originally triggered by a build-up of emotion/stress | <p>77. "I think the seizures were the build-up of it. I sort of feel it was just like a build-up, a mountain of things, and I got to the top and I got fed up of it..." (P9)</p> <p>78. "...if you don't get things off your chest, and you're holding certain things back, that keeps running round in your mind, then that is like stressing you out, then it's building up and making you more and more agitated, and I think that's possibly one of the causes that triggered it off." (P8)</p> |
|                                                                        | Relief after DS                                         | <p>79. "I used to feel exhausted (after a seizure), but it was almost like all that fear had gone, almost like a relief feeling, funnily enough, that it had gone." (P9)</p> <p>80. "I get a feeling as if something's subsided in me, I feel very calm again...." (P15)</p>                                                                                                                                                                                                                        |
| Inconsistent relationship between emotional triggers and DS occurrence |                                                         | <p>81. "At times when I'm stressed and fairly anxious I can have lots of seizures, but then at the same time I can have none at all..." (P12)</p> <p>82. "I can't see any pattern to it... so you could be happy, you could be sad, anything, it will just happen." (P8)</p> <p>83. "...before, I'd always feel like fear. I was just like really scared... now, I just feel like every kind of big emotion, just triggers it, that's the thing." (P7)</p>                                          |
| Post-seizure emotions / experiences                                    | Frustration                                             | <p>84. "So... they bring on a completely different emotion of 'Oh god, it's happened again' and 'I haven't done this and I haven't done that'... there can be a feeling of that, after one, which is 'Oh god, here we go, another day lost, another day wasted.'" (P2)</p>                                                                                                                                                                                                                          |
|                                                                        | Tiredness / exhaustion                                  | <p>85. "I feel very very tired, and sort of like, more worn out, then I was before..." (P11)</p>                                                                                                                                                                                                                                                                                                                                                                                                    |
|                                                                        | Weeping                                                 | <p>86. "When I come out of it, all I want to do is cry, and that's it... I'm always crying after them, I'm just not myself, if you know what I mean?" (P14)</p>                                                                                                                                                                                                                                                                                                                                     |

P = participant number



### **9.3.6. Relating to others**

Relationships with others were discussed frequently by patients. This cluster of themes included interpersonal sensitivity, caring for and supporting other people, and being a burden to others. These themes are illustrated in Table 44.

#### ***Interpersonal sensitivity***

Most patients reported being sensitive to other people's mental states. Some viewed this as a positive trait (statements 87 – 89), in that it allowed empathy for others and the ability to 'read people' well. However, some suggested that this interpersonal sensitivity could at times be a negative tendency because it could lead to misinterpreting social signals (e.g. statements 90 and 91), or that it might sometimes be better not to be so aware of others' mental states/emotions (statement 92). Some patients linked their interpersonal sensitivity to developmental experiences (e.g. statement 90).

#### ***Caring for and supporting others***

A prominent theme emerged relating to patients tending to be caring and supportive of other people, in a variety of ways. Several patients reported being the person that 'people come to with their problems', despite rarely sharing their own concerns and issues with others in return (statements 93 – 96).

#### ***Being a burden to others***

Linked to the above theme, several patients described their emotions or seizures as a possible burden on others, and invested considerable effort in avoiding disclosing their difficulties to others (e.g. statements 97 - 99). This was also connected to a need for independence and control, and viewing the self as strong and resilient (see subsequent section)

**Table 44. Themes about relating to others**

| <b>Theme</b>                            | <b>Sub-themes</b>                     | <b>Typical statements</b>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          |
|-----------------------------------------|---------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Interpersonal sensitivity</b>        | Interpersonal sensitivity as positive | <p>87. "I think I'm really good with other people...I do notice how people are feeling... I can stand back and think well what's made them feel that way?" (P15)</p> <p>88. "I can read people, even when you meet them, within, I don't know, a minute...and most of the time, I'm right." (P10)</p> <p>89. "I'm very sensitive to other people's feelings, and I have a lot of empathy." (P6)</p>                                                                                                                                                                                                |
|                                         | Interpersonal sensitivity as negative | <p>90. "I remember when I was younger...I was very susceptible to other people's emotions...I think having a fairly volatile home-life makes you that way. You're consciously looking out for stuff." (P4)</p> <p>91. "I take some people the wrong way, when they say something to me, I might take it the wrong way, even though they don't actually mean it that way...." (P8)</p> <p>92. "I'm going back to what I said about being sensitive to people, I just wish I didn't do that, I just wish I didn't understand it so well. Because sometimes, you just don't want to know..." (P2)</p> |
| <b>Caring for and supporting others</b> |                                       | <p>93. "...people always come to me. They talk to me about things, because I pick up straight away if something's wrong...I always put other people first, before myself." (P8)</p> <p>94. "I've never had no-one look after me, I've always looked after other people....but it's not like that now." (P3)</p> <p>95. "I've got time for, you know, even a complete stranger, to try to comfort them, whereas I ain't been comforted." (P11)</p> <p>96. "I think I have this thing where...in work...most people will come to me...I'm like an agony aunt. Which is ironic..." (P7)</p>           |
| <b>Fear of burdening others</b>         |                                       | <p>97. "I don't want to burden anybody else with it...I don't want to burden anybody else with what I've been feeling." (P3)</p> <p>98. "I tend to refuse people's help, I tend to not want to take anything." (P2)</p> <p>99. "...I'm really rubbish and I've always been quite rubbish about people supporting me, in whatever capacity. Be it emotionally or...I'm quite bad at that, and so to be reliant on anyone, I find very difficult." (P4)</p>                                                                                                                                          |

P = participant number

### **9.3.7. Resilience, protective factors and coping strategies**

Patients described factors that they perceived as being positive in their functioning or that facilitated coping with their difficulties. Some described having resilient personality traits and several patients recognised factors in their lives that were protective in some way. All patients described the use of coping strategies, adopted during times of emotional distress. Table 45 provides representative quotations.

#### ***Resilience***

Several patients described themselves as being emotionally ‘strong’, a ‘fighter’, tough and able to cope with adversity (statements 100 and 101). Some patients described this as a trait that had been learned early in life. Moreover, some perceived themselves as positive/optimistic, with a ‘glass half full’ outlook on life (statements 102 – 105). Several patients referred to a high need for control and independence (statements 106 – 108), and found that the occurrence of DS had significantly reduced their experience of confidence and autonomy in this regard (statements 106 - 108).

#### ***Protective factors***

A range of protective factors were mentioned, often viewed as positive aspects of patients’ lives. Spouses, family members and friends were discussed in positive terms by most patients, as sources of support and encouragement (statements 109 and 110). Additionally, some patients described academic and occupational success, also seeming to form a part of their sense of identity and self-confidence (statements 111 and 112). For some patients, work provided a relief from difficult life circumstances or emotions (statement 113).

#### ***Coping strategies***

The use of coping strategies was evident in most of the sample. These strategies most commonly involved attentional and behavioural distraction techniques, in addition to relaxation strategies such as meditation, martial arts, and enjoyment of music or nature (statements 114 – 118). A common, but less adaptive strategy was self-isolation and behavioural avoidance of social situations during times of acute emotional distress (statements 119 – 121), labelled ‘shutting down’ by several patients.

**Table 45. Themes relating to resilience, protective factors and coping**

| <b>Theme</b>              | <b>Sub-theme</b>                      | <b>Typical statements</b>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   |
|---------------------------|---------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Resilience</b>         | Emotional strength                    | <p>I 00. “I had to toughen up, to be strong...so I was a fighter, I fight for things that I want...” (P6)</p> <p>I 01. “I’ve always been able to cope with everything... because if you don’t help yourself, nobody will help you...” (P3)</p>                                                                                                                                                                                                                                                                                                                                                                                                                                                              |
|                           | Positivity                            | <p>I 02. “...if there is a negative, I try to turn it into a positive.” (P5)</p> <p>I 03. “...before I had all these things happen to me, I know that I was generally happy, a happy and outgoing person.” (P9)</p> <p>I 04. “I think I’m quite a positive person. I always look, I look positively on anything, I try to be optimistic, no matter what comes my way...” (P15)</p> <p>I 05. “I’ve always been more of a glass half full than glass half empty person....” (P9)</p>                                                                                                                                                                                                                          |
|                           | Control                               | <p>I 06. “He married a confident, incredibly confident, incredibly bossy, forceful, dominant woman. And I am a very very long way away from the person that he married now. I’m not that person now.” (P1)</p> <p>I 07. “...I was, I did everything. I was in charge and that was it, full stop. But now, I’ve lost it, I just haven’t got the confidence I had...With the seizures, since I’ve been having these turns...I was always independent, always. But I’ve lost it...I can’t cope with this, because it’s not me, I’m not in control, and I don’t like that.” (P3)</p> <p>I 08. “I don’t like losing control...in things which are related to me, it’s definitely out of the question.” (P10)</p> |
| <b>Protective factors</b> | Supportive relationships              | <p>I 09. “I know, even on my worst days, that fundamentally I am actually really lucky, and I’m actually very happy...I know that I’m lucky with my lot, and everything I’ve got in my world, my family, my friends, and everything...” (P1)</p> <p>I 10. “I’ve got people around me, so that helps you deal with things better and it makes you feel a little bit better, just knowing that people, you’ve got friends that’ll be there...” (P8)</p>                                                                                                                                                                                                                                                       |
|                           | Educational/occupational achievements | <p>I 11. “I did excellently at school, I made sure I did excellently at college, I did excellently in jobs...” (P2)</p>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     |

|                          |                                                  |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          |
|--------------------------|--------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
|                          |                                                  | <i>I 12. "...sometimes I just think ok yeah, stop complaining...you can have a job, some people don't have jobs, they don't have qualifications, or they are very sick and things like that." (P7)</i>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   |
|                          | Work                                             | <i>I 13. "...it's the one place that I always feel that I can be myself, but even if I don't feel well, if I go into (work), then I immediately feel well, because it's like a feel-good factor." (P15)</i>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              |
| <b>Coping strategies</b> | Distraction and relaxation                       | <i>I 14. "There are times when I try and distract myself, if I've realised that I'm feeling, you know, I'm having a bad day." (P1)</i><br><i>I 15. "I've had to sort of force myself to get out of the depressions and try to find things to do to take my mind off it....anything to take my mind off it." (P12)</i><br><i>I 16. "I might go over the beach for a while...that's nice, it helps, it don't get me completely calm, but it's a nice relaxing atmosphere and that's what I try and do, I try and put myself in a situation where I go and do something relaxing, and try and calm down." (P13)</i><br><i>I 17. "If I'm feeling down in the dumps, which I have been this week, I think right I'll just put my music on...it makes me feel better...cos I think of the good times that we had." (P3)</i><br><i>I 18. "...when I was young I did a lot of martial arts, so I learned how to do meditation and breathing exercises and things, and I use them now." (P12)</i> |
|                          | Behavioural / social avoidance ('shutting down') | <i>I 19. "If I'm going through a stressful time, then I shut down...so I, stop talking to friends...I just focus on what I need to try and do, and what have you. I stop answering text messages, and turn off my phone..." (P4)</i><br><i>I 20. "So I kind of withdraw, I don't phone call, I don't pick up my phone, I don't go out, I just stay in my room...it can go on for days...I tend to...shut down." (P7)</i><br><i>I 21. "...sometimes I have a tendency to shut down. You know, erm, switch off....you know, me mobile phone, and not answer the door....I mean, shut off from society, you know, where I won't come out, you know I'll I stay in...this is how I find my way of dealing with it." (P11)</i>                                                                                                                                                                                                                                                                |

P = participant number

## **9.4. Discussion**

### **9.4.1. Summary and interpretation**

The presently described study was conducted in order to gain further insight into the emotional experiences of patients with DS, and how these patients perceive their emotions to relate to the onset and recurrence of their seizures. The aim was to enrich and extend the quantitative findings described in previous chapters, to generate possible hypotheses for future research, and to provide an indication of areas of possible importance for clinical intervention. In order to achieve this, semi-structured interviews were carried out with a small subsample of patients who had completed the quantitative aspects of the research. The interviews focused on patients' general emotional functioning, how the seizures might relate to emotional states and/or difficult life events, and understanding emotions in others. The findings are discussed sequentially below.

#### ***General emotional functioning***

It is known that patients with DS tend to present with considerable emotional distress and difficulties in regulating emotional reactions, for example, depression, anxiety and borderline personality traits (e.g. affect dysregulation) are common in this population (see Chapter 2, sections 2.3.2. and 2.3.3). This study provides further support for the existence of altered emotional functioning in this patient group. It seems that patients with DS are most aware of frequent and intense states of negative affect, in addition to oscillating mood states (emotional lability) that may occur for specific reasons (i.e. in response to stressors), or that may be perceived as relatively unprovoked and rather random. In addition, several patients subjectively described (often intrusive) rumination on their affective reactions and the situations that provoke them. This is in line with a finding by Tojek et al. (2000) in which patients with DS were found to ruminate about previous life stressors to a greater extent than controls with ES.

Moreover, the intermittent episodes of excessive emotional expression described by patients seemed to occur when their perceived ability to cope with their emotions or stressors had reached a limit, in other words, when 'things had gotten too much'.

Difficulties in calming down once such episodes had been triggered were also described. It is interesting to consider whether DS may serve a similar function or to these episodic losses in emotional control, as another response to stress or negative emotions reaching a threshold (this idea was presented in Chapter 3, section 3.6). Nevertheless, the findings described above indicate that patients with DS experience considerable emotional distress, dysregulation, and at points, externalisation of this affect when it reaches unmanageable levels. These findings are in accordance with some of the quantitative results, in which depression, anxiety, affect dysregulation, and tension reduction activities, for example, were found to be elevated in the patients compared to the controls (Chapter 5, section 5.3.3). Moreover, the present qualitative findings indicate that patients also seem to show considerable insight into these emotional phenomena and so are not in outright 'denial' or ignorance of their psychological difficulties. Several patients directly attributed their emotional difficulties to the occurrence of DS, or felt that DS at least exacerbated their emotional problems.

It is particularly interesting that the above emotional problems were described alongside a general tendency to exert excessive control over the experience and/or expression of negative emotion. Phenomena described within this theme can accurately be summed up by the two phrases 'shutting down' negative emotions and 'putting on a brave face'. Basically, patients described a tendency towards inhibited experience of negative emotion, which could be conceptualised as a dissociative mechanism (i.e. emotional constriction, depersonalisation), in those patients who experienced this as relatively automatic and involuntary. For these patients, this type of detachment response seemed to have developed into a habitual tendency, possibly as a consequence of early life stressors such as familial dysfunction and/or parental modelling.

Some patients also avoided expressing genuine emotions to others, and/or referred to displaying an overtly positive façade even when this was incongruent with their actual emotional state. For some, this was a voluntary coping strategy, and seemed to be linked to beliefs regarding the necessity for complete independence, autonomy and personal control. These general patterns are reflective of the quantitative findings

pertaining to elevated scores on dissociative symptoms, such as emotional constriction and depersonalisation described in Chapter 5 (section 5.3.3). Moreover, this may also be related to the findings of Urbanek et al. (2014), that patients with DS report exerting greater control over states of anxiety and sadness, and more negative beliefs about emotions (e.g. shameful, irrational, useless), relative to healthy controls.

The strong links between emotions and bodily experiences described by some patients also suggested that negative affect was associated with an exaggerated focus on somatic symptoms and experiences. This is in line with the research findings that have shown high levels of somatoform symptoms in patients with DS (see Chapter 2, section 2.3.2), and the findings on somatoform dissociation presented in Chapter 5 (section 5.3.3). Together, these findings suggest that in patients with DS, difficult emotions and heightened stress may manifest as a variety of somatic symptoms (e.g. pain, gastrointestinal upset, sleep disturbance), and such symptoms may allow the individual to reduce the subjective experience of those unpleasant emotional states. The specific mechanisms by which emotion may give rise to somatoform symptoms is a fascinating and important area for further neurobiological studies.

### ***Stressful and traumatic life events***

The themes pertaining to stressful and traumatic life events, once again, generally reflected and extended the findings of previous studies (Chapter 2, section 2.2.1 and 2.2.2), and the results on adverse life events presented in Chapter 5 of the present thesis (section 5.3.3). The qualitative findings described here indicate that patients with DS experience a wide range of adverse life events and perceive their lives as currently and/or historically very difficult. The fact that abuse (physical, emotional, sexual) was disclosed by several patients further suggests the aetiological importance of these types of experience in DS, and shows that patients are generally aware of these experiences and the impact they have had. Several patients also described locking these experiences away, burying them or suppressing them, which indicates that the trauma and psychological consequences may not have been fully processed by these individuals. This has obvious clinical implications and is suggestive of a



complex and unresolved response to early traumatic experiences that may contribute significantly to the development of DS.

Relationship disturbances were also clearly present for many patients, particularly involving problems with significant others that appeared longstanding and difficult to resolve. Again, this is in accordance with previous quantitative studies, in which family/relationship dysfunction has been reported several times in this group (see Chapter 2, section 2.2.3). Several patients described dysfunctional family dynamics in childhood, with considerable insight into the possible psychological consequences of these relationships. Moreover, relationship crises and/or breakdown occurred immediately prior to DS onset in some cases. It is interesting to note that these reports conflicted with the fact that childhood family dysfunction was not observed when measured quantitatively (Chapter 5, section 5.3.3). It might be the case that the dysfunction described in the qualitative study was of a different nature to those aspects assessed using the Family Environment Scale, or that only a subgroup of patients experienced childhood family dysfunction.

Whilst there were other generally traumatic or adverse life events described in the current study (i.e. bereavement, occupational stress), which were often multiple, the consistent references to somatic complaints and medical illnesses highlights the important and distressing role that bodily-related experiences play for this patient group. The symptoms and occurrence of DS were described as a significant stressor that impacted on emotional functioning in the short- and long-term. Therefore, it is clear that DS has a negative impact on patients' stress levels and functioning. Moreover, the presence of other (possibly medically unexplained) symptoms is also important, as these were often long-term and seemed to cause considerable distress or reduced functioning. The most commonly reported complaints were pain and sleep disturbances. These symptoms could increase a tendency towards an attentional focus on somatic processes, in addition to causing increases in general distress (e.g. health-related anxiety).

Furthermore, sleep disruption is known to increase the likelihood of other physical and psychological symptoms, such as weight and appetite disturbance, depression and

increased subjective/physiological signs of stress. Therefore, it seems that sleep deprivation might exacerbate or contribute to the problems experienced by some patients with DS and should potentially be explored in further studies. However, as recently noted by Pavlova, Allen and Dworetzky (2014), sleep disturbances have not yet been studied extensively in this group.

It was interesting to note that, for some patients, these somatic complaints were attributed to negative emotions (i.e. anxiety, stress), and so it seems that these variables could be bidirectionally related. In other words, as with DS themselves, the experience of pain and sleep disturbances are likely to increase emotional distress, allocation of attention to bodily processes and physiological stress responses, but these responses are also likely to exacerbate the somatic symptoms, thereby creating a perpetuating cycle.

### ***Emotion and DS***

The study has also provided further evidence indicating that traumatic and/or stressful life events serve as precipitating factors for the initial onset of DS. The finding that almost all patients in this study could identify at least one stressful circumstance or life event at the time of DS onset indicates a fairly consistent pattern. Moreover, the wide range of events described indicates that precipitating stressors may take a variety of forms, including physical trauma (e.g. severe medical illness, invasive medical procedures), bereavements, occupational stress, relationship crises, and bullying. Again, these findings generally concur with findings from previous studies (e.g. Binzer et al., 2004; Bowman & Markand, 1996; 1999; Tojek et al., 2000).

Nonetheless, it is important to note that whilst most patients could identify stressors around the time of seizure onset, many did not portray a clear understanding of the ways in which such stressors might contribute to the occurrence of DS. Therefore, patients seemed to have limited insight into the processes by which their seizures had been triggered by the life events described. On the other hand, it was clear that some patients had a better understanding of how their emotional states might relate to the ongoing occurrence of DS. The theme of DS being a form of emotional release or 'shut-down' following a build-up of emotion was surprisingly insightful. These findings

are also reminiscent of those reported by Carton et al. (2003) and Wyatt et al. (2014), for example. Patients' perceptions of DS as a release or shutting down of emotion are in line with the commonality of ictal dissociative experiences (mental state symptoms of depersonalisation/derealisation) reported in Chapter 5 (section 5.3.3). Together, this lends preliminary support to the proposal that DS are dissociative phenomena (see Chapter 3, section 3.6). However, there are limitations to this interpretation (see below section 9.4.3), and so this must be seen as a tentative conclusion at present.

The inconsistency with which DS were associated with particular emotional states indicates that, if triggered by emotion, the relationship is not specific to any one emotional state (e.g. anger, fear), but rather that DS may be triggered by a variety of intense emotions. In addition, as many patients described DS occurring during times of relative happiness or relaxation (similarly to patients interviewed by Wyatt et al., 2014), it might be that the emotional 'shut-down' or 'release' does not necessarily occur in response to one emotional episode, but indeed might well occur after a gradual build-up and when the individual is in a 'safer' situation for the attack to occur. Either way, the lack of consistency most likely underlies the perceived unpredictability of the seizures and may make attempts at control or prevention more difficult. The perceived unpredictability of seizures expressed by the patients in this study concurs with previous literature that suggests that many patients feel that their attacks occur 'out-of-the-blue' (Reuber et al., 2011).

Emotions and experiences that were reported to occur after the seizures were somewhat more consistent and predominantly included exhaustion, frustration, weeping, and a sense of relief. It was apparent, therefore, that there were considerable emotional consequences of seizure occurrence in the short-term. This echoed the fact that patients also felt that there had been significant longer-term negative emotional consequences of the disorder. Again, there are clinical implications related to these findings, in that approaches incorporating seizure-related beliefs and representations (e.g. cognitive behavioural therapy) may be useful in assisting patients to develop more adaptive emotional responses to the occurrence of seizures, and thus reducing their emotional impact. Whilst this may not specifically

address the causal processes, such approaches could prevent worsening of emotional functioning and seizure occurrence.

### ***Relating to others***

The finding that patients generally described sensitivity to others' emotional states was particularly interesting, in the light of previous research and the experimental findings presented in this thesis. Patients' statements suggested that they were very much aware of and responsive to others emotions, which was seen as a positive tendency but also as negative by some. This accords well with the findings of Bakvis, Roelofs, et al. (2009) and those presented in Chapter 6 of the current thesis, that patients with DS show exaggerated vigilance for outward signs of others' emotional states (i.e. from facial expressions). Furthermore, the qualitative findings described here suggest that at least some patients are aware of this tendency and so it may not be operating entirely under the level of conscious awareness. Indeed, some patients were able to reflect on the origin of this vigilance, in difficult childhood family relationships.

Moreover, patients' subjective insights indicated that whilst this vigilance can at times be beneficial in their social relationships, for some it can also be detrimental, particularly when the social emotional cues are misread and the evaluation is inaccurate. Again, in the light of the quantitative findings presented in Chapter 7, this is particularly interesting. Those results suggested that patients with DS were generally less accurate than controls in reading facial expressions. Combined then, it seems that in this patient group, there is vigilant monitoring of others' emotions and some degree of 'jumping to conclusions' in interpreting others' expressions.

The theme regarding caring for and supporting other people may also be linked to the above pattern. Patients tended to describe themselves as the person that people turned to with their problems, as supportive and empathic individuals. It is possible that the vigilance to others' emotions may be facilitative of DS patients noticing signs of distress or negative affect in others' and thus, intervening and providing support when necessary. This is also likely to be associated with the tendency to appear outwardly 'strong' and resilient (see below) and to avoid burdening others with their

emotions. As such, it seems that the general tendency is that patients with DS appear to inhibit the experience/expression of their own emotions (particularly negative), whilst being attuned towards and responsive to those of other people.

### ***Coping, resilience and protective factors***

The fact that patients often referred to their resilient personality traits (e.g. being a fighter, optimistic, positive) suggested that they perceived themselves as proficient in coping with adversity and felt psychologically 'strong'. Again, this seemed to be closely associated to the high levels of control and independence preferred by these patients, and the ability to exert high levels of regulation over their emotions. In some cases, the resilience was seen as a natural response to the high levels of adversity that patients had experienced during their lives. However, for some patients, it was felt that the occurrence of DS had negatively affected these resilient characteristics and had left them less confident, in control and independent. This was clearly a source of considerable distress in some instances.

It is possible that an exaggerated emphasis on being resilient in the face of adversity and maintaining a relatively positive perspective may be crucially linked with the tendency to not tolerate the experience or expression of negative affect. Therefore, these beliefs and expectations imposed on the self could be an important mediator of the relationship between traumatic/stressful life events and the development of somatoform/dissociative symptoms, including DS. Put simply, patients' psychological resistance to processing and expressing trauma- or stress-related negative affect (i.e. the belief that acknowledging negative affect or accepting support is a weakness) could directly increase the tendency towards dissociation (psychological/somatoform) and thus, the development of DS or other MUS.

The coping strategies described in this study were also of interest. Several patients described using distraction techniques to cope with difficult emotions or situations, some of which appeared to be rather useful and adaptive strategies to allow them to feel calmer or less anxious, such as meditation, listening to music, the internet, or enjoying nature. However, the common reports of using behavioural and social withdrawal/avoidance indicated that some patients used strategies that are somewhat

less adaptive, and that could be detrimental to their general functioning. The excessive use of behavioural avoidance, for some, included prolonged periods of self-imposed isolation, which would necessarily lead to reduced socialisation, physical exercise, among other consequences. Once again, though, these reports are in accordance with previous literature which has indicated a tendency towards avoidant coping in this group (see Chapter 2, section 2.3.1). These findings suggest that this is an important area for clinical intervention.

Finally, the various protective factors described, such as supportive relationships, occupational and educational achievements, were also suggestive of areas for potential intervention in this group. It seems that, whilst patients with DS are very much aware of the stressors and traumatic events in their lives, some can also recognise areas in which they feel fortunate. As such, it may be that the presence of such protective factors limits the impact of DS, and may allow relatively better functioning than in those patients without them. Indeed, as seen in Chapter 1, findings have previously shown that outcomes tend to be better for patients with DS who are in employment (e.g. Duncan et al., 2011), those with more years of education (e.g. Reuber, Pukrop, Bauer, et al., 2003), and those who are accompanied by a supportive other to their first clinic appointment (Arain et al., 2007). It is possible that for patients out of employment, or with less education and positive social contacts, social interventions aimed at the development of occupational, educational and interpersonal skills might provide considerable benefits.

#### **9.4.3. Strengths and limitations**

This study had several strengths. Whilst quantitative studies have previously explored emotional functioning using self-report and experimental techniques, this is the first study to explore patients' phenomenological perspectives on these topics. The study provided insights into a number of important aspects of emotional functioning in patients with DS, from their own perspectives. Therefore, the study showed the extent to which patients were aware of their emotional strengths and difficulties, and provided rich information regarding how patients experience emotions and DS to be linked.

The findings of the study have a number of clinical implications, which if applied in practice, could allow interventions to target specific emotion-related processes in this group (e.g. emotion regulation, stress management, beliefs regarding emotional strength/resilience), in addition to seizure-related cognitions. Furthermore, the study has provided insights that could be used to generate testable hypotheses that could be examined in further quantitative study. The study also provided the patients included with an additional opportunity to be involved formally in a research programme seeking to address aetiological factors in the disorder, with more flexibility than quantitative techniques alone would allow. To date, most qualitative studies have focused on reactions to the diagnosis and treatment-related issues.

In terms of limitations, there are several that apply to this study as with qualitative studies in general. Obviously, the lack of inclusion of a control group in such a study precludes drawing absolute conclusions regarding the specificity of the findings to patients with DS. It is possible that some of the phenomena described may be present in other clinical disorders or in healthy individuals. Whilst this can be seen as a limitation of qualitative research, the goal of this study was not to draw firm causal conclusions regarding emotional functioning in DS, but rather to explore patients' understandings of such processes from their individual experiences, and to identify areas of possible importance for clinical interventions.

Moreover, another possible limitation is that this study aimed to examine patients' subjective and conscious experiences of the links between emotions and DS. According to most theoretical models of the disorder, the mechanism underlying this relationship is likely to occur below the level of conscious awareness. Indeed, the involuntary nature of the seizures is often underscored by patients generally perceiving their seizures to be beyond conscious control, and because they find it difficult to describe a specific relationship between emotional states and the occurrence of DS. Therefore, it could be argued that an attempt to examine patients' conscious access to these processes is unlikely to be informative. However, the study has shown that this sample of patients with DS were able to introspect and reflect on their general emotional functioning and processing styles, which in itself provides important insights into possible aetiological processes. Moreover, it is of considerable

interest that several patients were able to formulate a possible mechanism by which their emotions might be linked to their DS, in the form of an emotional release or shut-down. Therefore, it seems that at least some patients with DS are able to demonstrate insight into a possible emotional mechanism underlying their seizures. Nonetheless, it is clear that a consistent relationship between specific emotions and the onset of seizures is either not present, or is not discernible by patients themselves.

It should be noted, however, that an important possible influence on patients' responses in this study was any formulation provided by relevant clinicians. For example, all patients included in the sample had received a diagnosis of DS, and as such are likely to have received verbal and/or written explanations as to how the seizures might be related to psychological factors. In addition, some patients may have been directed towards internet-based resources for individuals with functional neurological symptoms. Therefore, it is not possible to conclude that the views expressed by patients in this study had not been in some way influenced by clinician's or other experts' theoretical viewpoints. Nevertheless, one of the exclusion criteria for recruitment for this study (as with the quantitative studies) was having undergone psychological interventions for DS, so it can be assumed that none of the patients included had experienced extensive therapeutic input that might have focused on seizure- or emotion-related beliefs or processes.

It is also important to note that within IPA, the investigator actively interprets the data provided by participants and as such, this interpretation may be in some ways biased by the sociocultural and professional background of that investigator. Indeed, whilst efforts were made to 'bracket' such influences when interpreting patients' responses, bias cannot be excluded. It is not possible to conclude that the research student was theoretically neutral at the time of the interviews or the analysis, having prior theoretical knowledge pertaining to DS and related processes (i.e. dissociation, trauma-related psychopathology). Therefore, it is possible that the interpretations provided in this chapter may have been influenced by this prior knowledge. Nevertheless, the research student made every attempt to 'stay close to the data'



and avoid making significant theoretical assumptions that were not consistently supported by patients' statements.

#### **9.4.4. Conclusions**

The current study has shown that patients with DS have considerable insight into their emotional functioning and how their life events and emotional responses might relate to the initial onset of DS, and ongoing seizure recurrence. Almost all patients could identify life events that may have contributed towards the initial onset of DS, or that were ongoing. The findings indicated the experience of considerable emotional distress and dysregulation that was often long-standing, but also exacerbated by the development of DS. Despite these difficulties, patients were generally aware of others' emotions and needs, and invested effort in caring for and supporting others. However, there was a clear tendency for the patients to highly value emotional resilience, control and independence, and to exert excessive levels of control over their experience and expression of negative emotions. These tendencies represent a psychological coping mechanism whereby short-term functioning and the appearance of 'coping' are maintained. This excessive emotional control, however, is not maintained consistently, and may contribute to episodes of excessive emotional dysregulation. Indeed, DS may represent a consequence of this unprocessed and 'bottled up' negative affect, allowing the individual to release or 'shut-off' from the resultant emotional experience. Future research might seek to test some of these hypotheses with experimental or self-report measures.

## **Chapter 10. General discussion**

### **10.1. Summary and interpretation of key findings**

The main findings are briefly summarised and interpreted individually in the subsections below. For parsimony, many of the exploratory findings are not discussed again in this section. Each section discusses how the key findings might relate to the models first proposed in Chapter 3 (section 3.6). Table 46 displays a summary of the aetiological variables supported by the findings presented in the thesis, and how they might contribute to the development, onset and/or maintenance of DS. The variables in bold are those that received the strongest support (i.e. experimental evidence, regression analyses) in the current research.

#### **10.1.1. Psychosocial factors**

##### ***Social/environmental factors***

The data generally supported previous findings and the hypotheses set out in Chapter 5. Patients with DS reported significantly more adverse life events and greater impact of these events, relative to the control group. More specifically, rates of sexual and physical abuse were elevated relative to controls, although no differences were noted for emotional abuse or neglect. Regression analyses suggested that the experience of sexual abuse was best predictive of a diagnosis of DS. The qualitative study also indicated the importance of remote traumatic experiences, particularly childhood abuse. However, a range of traumatic and/or stressful life events were disclosed by patients in the interviews, which were often multiple or prolonged. Other life events described included medical/somatic symptoms, diagnoses or crises, relationship conflict/disturbance, among other stressors (e.g. occupational stress).

These findings provide additional support for the importance of a range of previous adverse (traumatic/stressful) life experiences in predisposing to DS, although sexual abuse seemed to be a particularly important risk factor. This finding is consistent with the high rates of sexual abuse found in this patient group in previous literature (Sharpe & Faye, 2006).

**Table 46. Aetiological factors in dissociative seizures supported by the present research**

| <b><i>Predisposing</i></b>                                                                                  | <b><i>Precipitating</i></b>                  | <b><i>Triggering</i></b>                                                              | <b><i>Maintaining</i></b>                                                             |
|-------------------------------------------------------------------------------------------------------------|----------------------------------------------|---------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------|
| <b>Traumatic experiences (particularly physical/sexual abuse)</b> and heightened perceived impact of events | Elevated stress/distress levels (threshold)  | <b>Elevated autonomic responsivity to affective stimuli</b>                           | Relationship dysfunction/conflict*                                                    |
| Borderline personality characteristics                                                                      | Medical crisis                               | <b>Hypervigilance to emotional facial expressions</b>                                 | Psychopathology (PTSD, anxiety, <b>depression</b> )*                                  |
| Psychopathology (post-traumatic symptoms/PTSD, anxiety, <b>depression</b> )                                 | Traumatic events or trauma-associated events | <b>Reduced recognition and autonomic responsivity to emotional facial expressions</b> | Maladaptive cognitions (emotion-related beliefs)*                                     |
| <b>Somatoform dissociation/somatisation</b>                                                                 | Relationship crises / dysfunction            | <b>Somatoform/psychological dissociation</b>                                          | Dysfunctional coping strategies (social/behavioural avoidance)*                       |
| Psychological dissociation                                                                                  |                                              |                                                                                       | Borderline personality characteristics                                                |
| <b>Elevated autonomic responsivity to affective stimuli</b>                                                 |                                              |                                                                                       | <b>Elevated autonomic responsivity to affective stimuli</b>                           |
| <b>Hypervigilance to emotional facial expressions</b>                                                       |                                              |                                                                                       | <b>Hypervigilance to emotional facial expressions</b>                                 |
| <b>Reduced recognition and autonomic responsivity to emotional facial expressions</b>                       |                                              |                                                                                       | <b>Reduced recognition and autonomic responsivity to emotional facial expressions</b> |

\*these factors are likely to be bidirectionally related to DS occurrence

Bolded variables received the strongest support in the present thesis

Traumatic experiences in childhood and adolescence might influence mental health/psychopathology, personality development, emotional processing, and tendencies towards dissociation, and thus might interact with other predisposing factors in increasing the risk for DS. In addition, traumatic experiences might interact with pre-existing psychological vulnerabilities (e.g. cognitive deficits), in predisposing to the disorder. Traumatic and/or stressful experiences also seem to precipitate the onset of the disorder. The qualitative finding that almost all patients described one or more stressful/traumatic events around the time of seizure onset supports this hypothesis.

The quantitative findings regarding childhood family functioning, however, were discordant with previous research and the hypotheses of the study. The current sample of patients with DS rated their childhood family context similarly to controls. No differences were apparent for any of the domains examined, including childhood family control, expressiveness, conflict, cohesion, or independence. Therefore, it seems that childhood family dysfunction is not a universal or key causal factor in DS, although it might moderate the effects of other childhood traumas. Nevertheless, several patients disclosed difficult relationships or dynamics with one or more family members during childhood, although this was often in the context of other, more secure and adaptive attachment relationships. In the qualitative study, several patients referred to protective factors in their lives, such as work-related and/or educational success/achievements and supportive relationships with family members, romantic partners, or friends. These protective factors may moderate the effects of the risk factors and might influence outcomes in the disorder.

### ***Psychological factors***

#### ***Psychopathology***

In line with the hypotheses and previous literature (See Chapter 2, section 2.3.2), patients with DS reported elevations in depression and anxiety relative to controls, suggesting heightened levels of emotional distress. Subjectively, patients described several emotional difficulties including episodes of intense negative affect (often described as depression), either currently or in the past. Furthermore, DS patients

with some degree of traumatisation (i.e. rating one or more life event as having had at least moderate impact), reported significantly elevated post-traumatic symptoms relative to traumatised controls, across all symptom types (i.e. arousal, avoidance, re-experiencing, total). Moreover, 66.7% of the patient sample met criteria for a diagnosis of post-traumatic stress disorder (PTSD) according to DSM-IV (data were collected prior to DSM-5 publication). In the qualitative study, several patients discussed the impact that previous trauma (particularly in childhood) had had on their emotional functioning. These findings highlight the possibility that DS are associated with a complex and pathological psychological response to traumatic life events.

For some, these difficulties (e.g. depression, anxiety, trauma-related distress) predated the onset of DS and so might have acted as a predisposing or precipitating factor. In contrast, for others, depressive symptoms seemed to have developed after the seizure disorder, suggesting that DS might have triggered or caused the negative affective state. In such cases, once triggered, depressive symptoms might perpetuate DS, by increasing emotional distress and general dysfunction.

#### *Psychological and somatoform dissociation*

The current research provided further evidence for elevated psychological and somatoform dissociative tendencies in patients with DS, and supported the hypotheses presented in Chapter 5 (section 5.1.2). The use of a dimensional measure of psychological dissociation (Multiscale Dissociation Inventory) allowed the examination of a range of dissociative experiences, with the patient group receiving higher scores on all subscales (relative to controls), including Depersonalisation, Derealisation, Emotional Constriction, Memory Disturbance, Identity Dissociation and Disengagement. Moreover, somatoform dissociation was also significantly elevated in the patient group compared to controls, as measured with the Somatoform Dissociation Questionnaire.

A tendency towards reduced/inhibited experience and expression of (negative) emotions was also apparent in the qualitative findings, labelled 'shutting off'. Patients also expressed strong links between emotional states and physical problems (e.g. pain, sleep disturbance) or experiences (autonomic arousal). These phenomenological

findings seem to relate to dissociative mechanisms whereby unwanted emotional experiences are dissociated from conscious awareness in order to reduce their impact. These mechanisms seem to represent the way in which, at least some patients with DS, cope with their emotions in general. However, these dissociative processes might also represent the underlying mechanism by which DS are triggered (see Chapter 3, Figure 1).

#### *Borderline personality features*

The results on the Inventory of Altered Self Capacities suggested that patients with DS display elevated behavioural and psychological tendencies that would be associated with BPD. These included elevations (relative to controls) in concerns about abandonment in important relationships, affect dysregulation, tension reduction activities, and identity disturbances. Interestingly, the regression analyses in the current study indicated that, of the variables entered, scores on the subscale measuring affect dysregulation were best predictive of a diagnosis of DS. The qualitative findings also indicated that emotional dysregulation (alternating moods, emotional outbursts, difficulties reducing emotional arousal) was particularly problematic and/or distressing for most patients.

These findings support those of previous studies (e.g. Reuber, Pukrop, et al., 2004). Therefore, it seems that affect dysregulation (and/or other borderline traits) might be an important predispositional factor for DS. These characteristics could be central to the emotional distress experienced by patients with DS, and might, therefore, increase the risk of developing dysfunctional ways of coping with such distress (e.g. externalising or avoiding intense negative affect, somatisation/dissociation). However, it is also suggested here that such personality traits might perpetuate the disorder, by contributing to emotional disturbances, dysfunctional coping and relationship problems on an ongoing basis, and therefore maintaining distress.

#### **10.1.2. Emotions and dissociative seizures**

In the qualitative interviews, most patients could not identify any one emotional trigger for ongoing DS, suggesting that emotional triggers might involve generalised arousal rather than specific affective states per se. Nonetheless, a few patients

explicitly described their seizures as a way of releasing or 'shutting off' from intense emotional states. It was clear, however, that patients often experienced a range of negative emotional states following the occurrence of DS, suggesting that the seizures have a negative short-term impact on emotional functioning.

The quantitative findings pertaining to ictal symptoms partially supported those of previous studies (e.g. Goldstein & Mellers, 2006). The most common ictal symptoms were those of autonomic arousal (AA; i.e. racing heart, sweating), mental state (predominantly reflecting depersonalisation/derealisation), and cognitive symptoms (cognitive manifestations of anxiety/fear). Goldstein and Mellers (2006) reported that ictal AA symptoms were significantly more common in patients with DS relative to those with ES, which is consistent with the commonality of these symptoms in the sample included in the present research (most recent seizure = 88.9%; most severe seizure = 91.7%). This supports the hypothesis that DS are associated with elevated sympathetic ANS activation, as proposed by Goldstein and Mellers (2006) and in the model presented in Chapter 3 (Figure 1). However, the findings also extended those of the previous study, by showing that cognitive and mental state symptoms were also reported by a considerable proportion of patients (80-100%).

These findings suggest that during (or around the time of) DS, patients may experience considerable subjective anxiety (cognitive symptoms), in addition to feelings of detachment from the environment or themselves (mental state symptoms). Moreover, the significant correlations between dissociation scores (depersonalisation/derealisation) and ictal mental state symptoms further suggested a clear link between dissociative states and seizure symptoms. It is possible that the cognitive and autonomic symptoms of anxiety trigger a dissociative response, manifesting in the seizure symptoms and psychological dissociation. This hypothesis is discussed further below, with reference to the proposed model of DS.

### **10.1.3. Emotional processing**

#### ***Preconscious facial expression processing***

The main findings from the emotional Stroop experiment (Chapter 6) were that, after controlling for anxiety, depression and education, patients with DS displayed an

elevated attentional bias towards preconsciously processed emotional faces (angry/happy), relative to healthy control participants. Taken together with the findings of Bakvis, Roelofs, et al. (2009), this study provides additional support for the hypothesis that patients with DS show altered processing of others' emotional facial displays, when these displays are not readily available to conscious awareness. Whilst Bakvis et al. reported a specific effect with reference to angry faces, the current findings suggested that the effect was more generalised, including both happy and angry faces.

The findings are tentatively interpreted as a preconscious hypervigilance to alterations in the emotional states of other people. Interestingly, several patients described being highly aware of and attuned to others' emotional states (or changes in them), suggesting some degree of insight into this tendency. Moreover, a proportion of the sample reported some awareness of the facial stimuli in the emotional Stroop test; therefore, the preconscious nature of this bias is questionable, in the present study. In addition, it was not possible to exclude the possibility that medication use (i.e. AEDs) may have influenced the findings presented in Chapter 6.

Nonetheless, if such a hypervigilance is replicated in other studies, it would suggest that this bias is an important feature of the emotional processing style of patients with DS. As such, it might contribute to elevated levels of emotional distress and arousal in patients with DS, due to (preconscious) processing of potentially irrelevant emotional signals in others. Moreover, this hypervigilance might cause interference with other important psychological processes, as suggested by the findings of Gul and Ahmad (2014), for example. This interference could disrupt functioning in other domains, including cognitive processing and social behaviour. As such, this bias might act as a predisposing or perpetuating factor in DS. Furthermore, these preconscious processes might contribute to triggering individual DS, by contributing to elevated emotional distress and/or arousal, possibly without patients' awareness of the reasons for these feelings. These possible processes are discussed further below.



### ***Conscious facial expression processing***

The key finding described in Chapter 7 was that when patients explicitly attended to facial expression stimuli, they displayed reduced recognition accuracy. This effect was independent of depression, anxiety, education, general face processing abilities and medication use (i.e. AED or antidepressant use). However, there were no group effects for intensity ratings, suggesting that patients could accurately determine the level of emotional intensity in emotional facial expressions. The deficit was, therefore, specific to determining the emotional meaning of the expressions. Moreover, reduced autonomic responses (SCRs) to these stimuli were also observed in a subgroup of participants, relative to controls. These findings are in contrast to some patients' subjective reports of being sensitive to and adept at understanding the emotional states of others, in the qualitative study. However, it is interesting that some patients acknowledged that at times, they misinterpreted others' emotional states.

The deficit in determining the emotional meaning of facial expressions might be a predisposing factor in the development of DS. One possible explanation could be that developmental trauma (e.g. abuse, relationship disturbances) might disrupt emotional learning processes, whereby particular facial expressions would usually become associated with specific behaviours in others and/or particular emotions in the developing individual. Disruptions to this process might lead particular expressions to be associated with the 'wrong' emotional states in the self or others. This tentative hypothesis was suggested by the exploratory finding that recognition accuracy for neutral faces was negatively correlated with trauma scores. Despite such a hypothetical disruption, it is possible that the level of expressed/provoked emotional intensity could be learned appropriately, just in connection with the wrong emotional state (this would explain the lack of group differences in intensity ratings).

On the other hand, the reduced recognition and autonomic responding to facial expressions might represent a form of inhibitory, avoidant or dissociative response to them, which would serve to limit their potential psychological impact. Again, such a tendency might have developed in response to being exposed to unusually frequent negative or distressing facial expressions in others. The reduced SCR amplitudes to

the facial expressions might reflect some form of habituation process; whereby patients' have become desensitised to the emotional significance of others' facial expressions. Regardless of the specific cause of the deficit, reduced recognition and somatic responses to emotional facial expressions are likely to be associated with considerable distress and disturbed social functioning. As such, these tendencies could also perpetuate DS on an ongoing basis, and/or trigger individual attacks (see below for further discussion).

### ***Emotional responding to consciously perceived affective images***

Contrary to the hypotheses of the study presented in Chapter 8, no between-groups differences were observed in subjective emotional reactions to general affective images. Whilst the findings regarding valence were consistent with those of Roberts et al. (2012), the negative findings for arousal ratings were not. Roberts et al. reported elevated arousal ratings for positive and neutral images, relative to controls. It is likely that methodological factors might have accounted for such differences. The findings of the current study suggested that patients with DS experienced similar valence (positivity-negativity) and emotional arousal (low-high) as healthy individuals, in their subjective responses to the images.

However, in a subgroup of participants (autonomic 'responders'), the DS group showed elevated SCRs relative to controls, and this did not seem to be specific to one particular category of stimuli. This latter finding was independent of the influence of depression, anxiety, and medication use (AEDs, antidepressants). This finding suggests that on a physiological level, some patients with DS 'over-react' to affective images, although these physiological responses do not influence subjective emotional experience. This finding suggests a dissociation between physiological and subjective elements of emotion, in this subgroup of patients.

This tendency towards elevated somatic manifestations of emotion might predispose to developing DS in some patients. In addition, this pattern of responding might perpetuate the disorder, by contributing to ongoing elevations in general affective arousal. By doing so, such responses might maintain an arousal level that is closer to the threshold at which DS are triggered than would otherwise be the case.

Importantly, elevated autonomic reactions to specific affective stimuli (in the presence or absence of subjective responses) might then trigger individual seizures, by causing arousal levels to reach such a threshold. As proposed by Baslet (2011), this threshold is likely to vary between patients. Moreover, the degree of autonomic responsivity (and subjective awareness) is also likely to vary between cases. It is likely that this explanation might only apply to a subgroup of patients with DS. Nonetheless, this finding has considerable significance for understanding the mechanisms by which DS might occur.

### ***Summary and conclusions - emotion, DS and an affective-dissociation model***

Together, the findings presented in the thesis support several of the propositions made in Chapter 3 (section 3.6). In brief, the supported propositions are that DS are dissociative phenomena (i.e. involving a loss of voluntary control and awareness of psychological and/or somatic processes), and that emotional processing abnormalities may serve as predisposing, perpetuating and/or triggering factors in this disorder. Furthermore, the suggestion that elevated arousal levels may trigger individual DS occurrence is also tentatively supported.

Regarding the dissociative nature of DS, the thesis has presented evidence that patients with DS experience a range of dissociative symptoms, including psychological and somatoform manifestations. These experiences appear to be elevated in general daily life, but importantly, patients report dissociative phenomena occurring immediately before and during their seizures. Indeed, some patients articulated a clear understanding that their seizures serve the purpose of allowing them to detach (i.e. dissociate) from strong and intense emotional states. Therefore, in terms of the model presented in Chapter 3 (Figure 1), the box in which ‘dissociation’ is postulated has received support from the research presented.

In light of the findings, the model has been modified (see Figure 15) by the addition of the word ‘acute’ to the ‘dissociation’ box. This highlights the fact that the dissociative experiences occurring during DS represent a paroxysmal, time-limited state of acute dissociation. Furthermore, an additional box has been added to the model, in which ‘trait’ dissociation is specified. This illustrates that more general

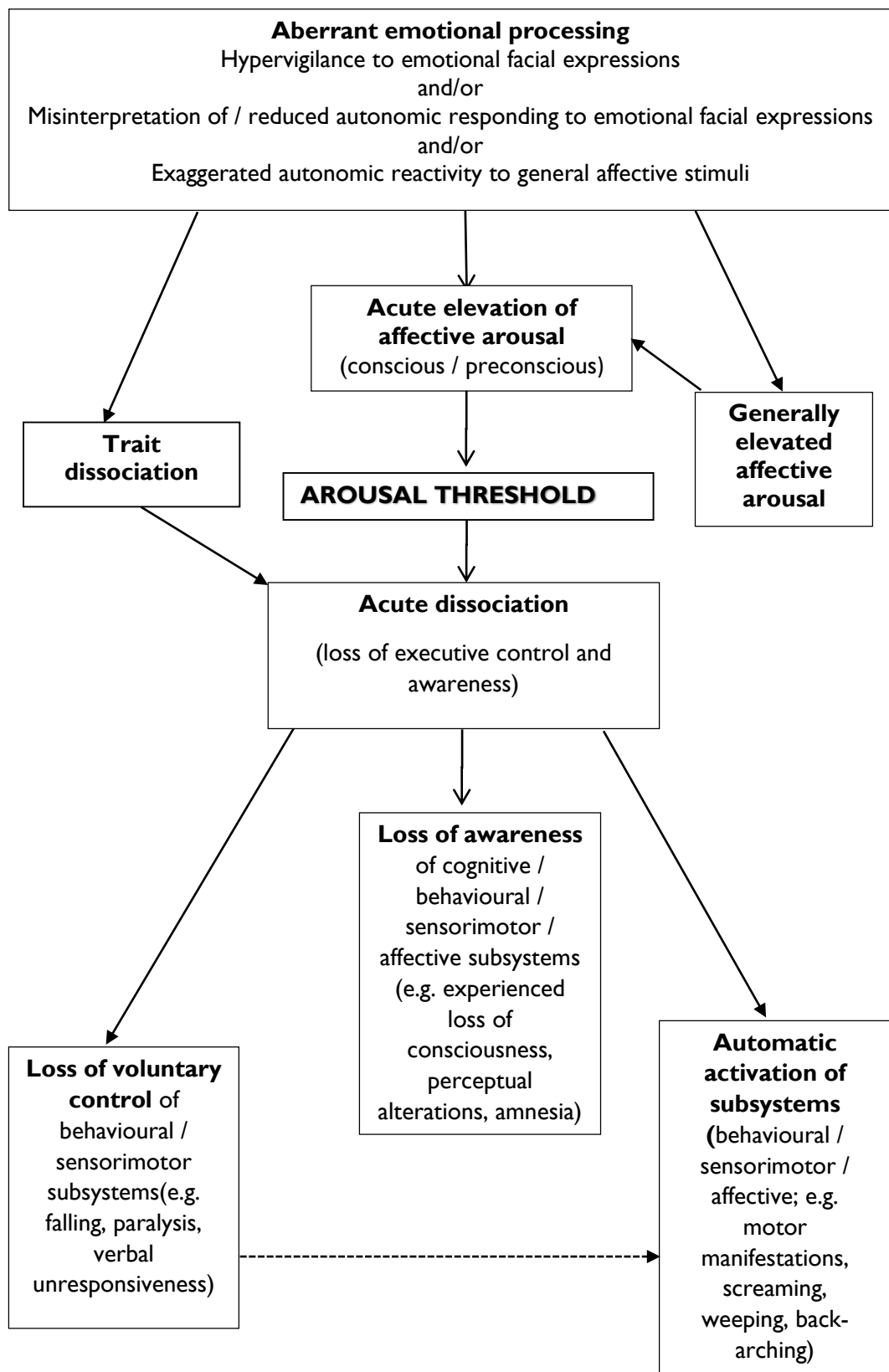
dissociative experiences occurring in daily life (i.e. not peri-ictally) may also be present in patients with DS (i.e. depersonalisation, derealisation, emotional constriction), and might increase the likelihood of acute dissociation (i.e. a DS), by perhaps 'rehearsing' dissociative responses to aversive psychological/somatic states. It is also possible that these general tendencies towards dissociation are influenced by aberrant emotional processing.

Turning to emotional processing biases, the findings have provided further evidence for the existence of a preconscious attentional hypervigilance towards social emotional signals (facial expressions) in patients with DS. In relation to the proposed affective-dissociation model, the finding informs the top level, in that it represents an aberrant emotional processing bias that might contribute to an acute increase in affective arousal in some circumstances (e.g. when signs of threat are perceived).

Moreover, this hypervigilance might also contribute to generally elevated affective arousal on an ongoing basis, which in turn would increase the likelihood that an acute rise in arousal would meet the 'threshold' at which DS might be triggered. In accordance with this, an additional box has been added to the model, labelled 'generally elevated affective arousal'. The box previously labelled as 'elevated affective arousal' has, therefore, been renamed 'acute elevation of affective arousal' to specify that this triggering arousal is a discrete, time-limited and acute increase.

When processed at a conscious level, patients with DS showed inhibited autonomic responses and impaired recognition of the emotional meaning of facial expressions. Combined with the hypervigilance described previously, these tendencies could lead to a situation in which the individual is overly alert to the emotional states of others, yet fails to accurately identify these when explicitly trying to do so. Regarding the triggering mechanism originally outlined in Figure 1, it is possible that subjective misinterpretation of others' facial expressions could contribute to generally elevated affective distress and arousal, by causing ongoing difficulties in social interactions and/or distress in response to misperceived hostility, for example.

**Figure 15. Modified affective-dissociation model of a DS episode**



In some circumstances (e.g. interpersonal conflict), the consequences of ‘misreading’ others’ facial expressions could create an intense/acute increase in emotional distress and/or arousal. Therefore, the deficit in facial expression recognition might contribute directly or indirectly to triggering individual DS. As such, this finding also informs the top level of the model displayed in Figure 15.

However, it is slightly more difficult to interpret how the observed reduced autonomic responding to facial expressions would relate to the proposed triggering mechanism outlined in the model. One possible interpretation of the findings is that attenuated SCRs in response to facial expressions in the short-term (e.g. seconds) could contribute to the observed deficit in conscious recognition, which in turn might give rise to elevations in emotional distress and arousal in the longer-term (e.g. minutes/hours). These processes require further examination; however, reduced autonomic responding to facial expressions seems to be characteristic of at least a subgroup of patients with DS, and so it has been added to the top level of the model in Figure 15.

Finally, the pattern of findings in the affective picture task suggested that some patients with DS generally respond to affectively significant stimuli with exaggerated physiological arousal, but that this arousal does not necessarily manifest in alterations in subjective experience. This could be described as a dissociative emotional processing style and might result in an accumulation of (somatic) emotional arousal that is not fully perceived by the patient. This response bias has, therefore, also been added to the top box in the model. As such, it could contribute to generally elevated affective arousal, thereby increasing the risk of reaching the hypothetical threshold at which DS might occur. However, when patients are faced with a specific stressor or adverse situation (e.g. interpersonal conflict, trauma reminders), the tendency to autonomically ‘over-react’ might serve as the ‘acute elevation in affective arousal’ which reaches the threshold and thus triggers the dissociative episode (the initiation of a DS).

It should be noted that the current research did not inform the level of the model in which an ‘arousal threshold’ was proposed. Whilst the qualitative findings indicated

that some patients saw their seizures as a culmination of built up emotion, the quantitative studies were not designed to test the hypothesis that a threshold is required for the initiation of a DS. Future studies might seek to examine this hypothesis more explicitly. Nevertheless, the concept of a threshold has been retained in this model, in order to explain the paroxysmal nature of DS.

## **10.2. General strengths and limitations**

This section summarises the general strengths and weaknesses of the research described in the thesis; however, the merits and limitations of the individual studies are not repeated here.

### **10.2.1. Strengths**

A key strength of the research presented is that it is the first attempt to examine several different aspects of emotional processing in the same sample of patients with DS. Whilst there have been previous studies of emotional processing in the literature (see Chapter 3, section 3.5), this is the first study to assess responsivity to social and more general affective stimuli in the same group of patients. Indeed, the study reported in Chapter 7 is the first of its kind in this clinical group. Moreover, the combined use of quantitative and qualitative techniques allowed an assessment of the extent of patients' subjective insights into these processes.

The measurement of relevant psychosocial variables (Chapter 5) also allowed for exploratory analyses of potential relationships between emotional processing biases and these variables. In addition, it was possible to control for potential confounding variables (e.g. depression, anxiety) when examining group differences on the experimental tasks, which allowed a more rigorous analysis of emotional processing biases specific to patients with DS. Furthermore, the inclusion of relevant standardised cognitive tests in the battery was an additional advantage, in that it was possible to determine whether the two groups showed equivalent performance on tests of cognitive abilities that may have affected performance on the experimental tests. Where the groups did not perform at a similar level, it was possible to determine the extent to which any influence of the relevant variable occurred.

The inclusion of a DS sample (n=40) larger than that used in several previous studies is also an advantage. Previous studies have tended to include samples of between 10 and 20 patients (e.g. Bakvis, Roelofs, et al., 2009; Roberts et al., 2012), although some investigators have recruited larger samples (e.g. Gul & Ahmad, 2014; DS n = 72). The inclusion of 40 patients allowed adequate statistical power to detect between-groups differences with covariates included in the analysis, and for the exploratory correlational and regression analyses. Moreover, the patient and control samples were generally well-matched on important characteristics that might have otherwise affected the findings obtained, including gender, ethnicity, and age. The exclusion of potentially confounding medical and psychiatric diagnoses from both samples also allowed a more precise examination of any differences specific to DS only.

A strength of the statistical methods used was that stringent alpha levels and/or post-hoc corrections were applied in determining the significance values, when multiple tests were conducted. This consideration ensured that the probability of Type I errors was minimised, and that the findings reported were more likely to reflect genuine effects rather than spurious findings relating to elevated familywise error.

### **10.2.2. Limitations**

A key limitation of the research was the use of a cross-sectional between-groups design, which can create difficulty in interpreting the direction of causality for significant effects. For example, whilst Chapter 7 showed between-groups differences in facial expression recognition between groups, it was not possible to conclusively infer whether the deficit in recognition in the DS group contributed to the disorder, or whether the disorder caused the differences. Other types of research design can improve the confidence with which conclusions can be drawn (i.e. prospective longitudinal designs).

Another potential limitation is the lack of a clinical comparison group. Whilst it was considered important to include a healthy control group in order to assess relative differences to normative performance on the measures included, an additional clinical control group might have provided further depth to this study. Possible control



groups are discussed below in section 10.4. The main problem with comparing performance of patients with DS to healthy controls is the fact that DS is confounded with medication use, the experience of chronic seizures, and/or loss of psychosocial functioning due to the seizures (i.e. social, occupational and educational constraints). Whilst the inclusion of a control group of patients with ES would rectify these issues, this would not be an appropriate control group due to the potential for seizure-related neurological issues (e.g. medial temporal sclerosis) to influence affective processes in that group (e.g. Meletti et al, 2009).

Related to the above is the fact that it was not possible to unequivocally exclude the possible influence of medication on the performance of the patients on the emotional Stroop test. As discussed in Chapter 6, it is known that AED use can be associated with cognitive side-effects (e.g. Aldenkamp et al., 2003) and mood alterations (e.g. Nadkarni & Devinsky, 2005), which could potentially have influenced the findings presented. However, several measures were taken to assess this possible source of influence. Various cognitive abilities (e.g. memory, general intellectual functioning, general face processing) and emotional distress (e.g. anxiety, depression) were measured and controlled for statistically (where differences were identified). Moreover, examination of absolute reactions times in Chapter 6 indicated that processing speed was not significantly slower in the DS group. In addition, it has been reported that the number of AEDs taken by patients with ES had no influence on facial expression processing in that group (Meletti et al., 2009). In fact, these authors found that the only AED to have a possible effect on facial expression processing was phenobarbital (which none of the patients in the present sample were taking at the time of testing).

However, it is known that the use of antidepressants (SSRIs in particular), might affect emotional/facial expression processing and its neural correlates (Arce et al., 2008; Browning et al., 2007; Harmer et al., 2003; 2006; Merens, van der Does, & Spinhoven, 2007). The literature on the effects of serotonin manipulations is complex; however, effects are often apparent in both healthy and clinical populations, such as enhanced recognition of facial expressions. Whilst the influence of antidepressants was excluded in Chapters 7 and 8, it is possible that some influence

was apparent on the emotional Stroop test in the present research. Future studies should include larger sample sizes of patients, with sufficient numbers of medicated and unmedicated individuals, thereby allowing a comparison of the possible influences of medication on these processes.

An additional possible weakness in the research presented here was that the diagnosis of DS was not based on video-EEG for all patients in the sample. The gold-standard in diagnosis of DS is now considered to be video-EEG, which provides a fairly conclusive test of the nature of the habitual seizures experienced by patients. In this study, 67.5% of patients had undergone video-EEG as part of the investigations into their seizures, with 90% having undergone routine EEG and 80% reporting at least one MRI scan. Altogether, these rates suggest that misdiagnosis would not have been a substantive issue in this sample. However, whilst unlikely, there is a small possibility that at least some of the patients included here may have suffered from comorbid current or previous epilepsy. An implication is that possible comorbid ES may have affected the measures of emotional processing (e.g. Meletti et al., 2003). However, this limitation would also apply if 100% of the patient sample had undergone video-EEG, as an absence of epileptiform activity in the monitoring unit does not provide absolute certainty that the individual does not also experience some ES. Therefore, this limitation is one that must be considered when examining any study of this nature in this patient group.

Furthermore, all patients were recruited from specialist neuropsychiatry clinics. Not only would these patients be more likely to be complex cases and/or presenting with psychiatric comorbidities, all patients would have accepted a referral to mental health services. This represents a potential source of bias in the sample. Moreover, regarding seizure semiology, patients were not categorised into subgroups on the basis of seizure 'types' (see Chapter I, section I.3.2). It may have been beneficial to examine subgroups of patients on this basis, in order to examine whether any of the psychosocial or emotional processing variables were associated with particular presentations. In the current research, the sample size precluded such an analysis.

An additional limitation of this study was that the groups were not sufficiently matched for years of education (YoE). Whilst attempts were made to match this variable during recruitment, the final sample differed significantly. Whilst YoE was controlled for statistically within the data analyses, this is not a desirable difference between experimental groups. For example, within some of the regression analyses, the effect of YoE remained significant when other factors were held constant, suggesting that this factor was an important predictor of group status. Despite this difference, the groups did not differ on general intellectual functioning (IQ) scores; therefore, the difference in education does not seem to have been associated with considerable differences in cognitive functioning.

Statistically, some authors have argued that it is not appropriate to control for differences in variables that vary systematically between two populations (e.g. depression or anxiety in this instance), using statistical corrections. For example, Miller and Chapman (2001), argued that ANCOVA is widely misused to this effect, particularly in psychopathology research. However, whilst this argument may hold for some variables, it is not the case that differences in anxiety and depression necessarily reflect a substantive difference between patients with DS and healthy controls. There are many healthy individuals who experience mild to moderate symptoms of anxiety and depression, and likewise there are many patients with DS who do not. Moreover, as discussed above, it is just as likely that depression and anxiety are a consequence of the seizures as that they contribute to the causation of DS. The aim of the research here was to determine the potential emotional processing biases specifically associated with DS, and so the decision to control for symptoms of common psychopathology was made. Future studies might seek to manage this issue in alternative ways, such as including control groups of individuals with subclinical or clinical levels of depression or anxiety but without DS (or any other dissociative or conversion disorder).

### **10.3. Possible implications of the current research**

The current findings have considerable implications for the understanding and treatment of DS. In terms of theoretical implications, a better understanding of emotional responding in this group provides important insights into which individuals

are most at risk of developing the disorder, the mechanism by which individual seizures are triggered, and possible emotional processing biases which might contribute to perpetuating the seizures. As discussed previously, negative and/or trauma-related emotions have been seen as a core cause of DS and conversion/dissociative disorders for some time. However, the empirical literature has generally not sufficiently examined emotional processes in DS as yet. Whilst work has started to be published in this area, there are still many questions to be answered. The research presented here was an attempt to answer some of these questions, and to indicate important avenues for further experimental research.

On a practical level, the most important implications are those pertaining to clinical issues such as diagnosis and treatment of patients with DS. Given that DS seem to be associated with alterations in emotional processing, avenues for relevant interventions might involve the development of stronger emotion recognition skills, or might be aimed at reducing attentional distraction by emotional stimuli. These could be carried out using computerised training packages, for example. Cognitive interventions such as cognitive bias modification (Koster, Fox, & MacLeod, 2009), utilise experimental methodologies to attempt to modify dysfunctional biases in other clinical disorders (e.g. depression, anxiety; Hallion & Ruscio, 2011). Perhaps similar techniques could be applied to the specific biases present in patients with DS (which would require further elucidation in research studies first).

Moreover, a suitable clinical approach might focus on integrating awareness of subjective and somatic emotional experiences. Importantly, techniques could perhaps aim to develop patients' awareness and tolerance of somatic emotional arousal. Interventions aimed at managing, reducing or releasing affective/somatic arousal may be particularly relevant (e.g. relaxation, meditation, physical exercise). Whilst some of these approaches might currently be incorporated into other treatment models (i.e. CBT), some patients might benefit from more focused work in these areas. Vancini et al. (2014) have recently advocated the recommendation of physical exercise programmes for individuals with DS.

Interventions might seek to explore emotional awareness and processing more

generally, aiming to improve patients' willingness and ability to tolerate and work through negative affective states, rather than avoiding or dissociating during them. Indeed, Howlett and Reuber (2009) incorporated aspects of emotional processing in their integrated treatment approach for patients with DS. Mindfulness-based therapies may well be useful for patients with DS (Baslet, 2011; 2012; Baslet et al., 2014; Baslet and Hill, 2011). Mindfulness-based therapies could facilitate the allocation of attentional resources to possible triggers and internal states, allowing patients to gain more insight into the series of events that serve to initiate their seizures (Baslet, 2011).

Psychoeducation aimed at informing patients about the types of symptoms that might be dissociative might be beneficial, so that patients can identify such symptoms and then possibly redirect attention to their emotional precipitants, in order to develop more adaptive coping responses. Indeed, psychoeducational websites and pamphlets are becoming more common in 'first-step' treatment of DS (e.g. Mayor et al., 2013; N.C.Thompson et al., 2013).

Moreover, given the findings presented in this thesis, another possibly useful approach might be the use of mentalisation-based therapies, as have been applied with patients with BPD (e.g. Bateman & Fonagy, 2004; 2006). Mentalisation-based therapies aim to improve patients' ability to consider the mental states of the self and others, and how these might relate to behaviour. As such, these techniques might be particularly beneficial for interventions with patients diagnosed with DS who report similar difficulties as patients with BPD (e.g. affect dysregulation, interpersonal conflict, tension reduction activities). Moreover, the evidence for impaired facial expression recognition in the current sample (Chapter 7) also indicates the possible value of this type of approach.

Relating to the findings on traumatic experiences, it is possible that interventions might seek to explicitly address trauma-related distress. Treatments for PTSD have increasingly focused on facilitating emotional processing of the traumatic memories, with evidence to suggest that techniques such as prolonged exposure and EMDR are useful in reducing PTSD symptoms (e.g. Bisson et al., 2007; Rauch, Eftekhari, & Ruzek,

2012). Perhaps, in patients who disclose significant traumatic histories or who meet criteria for a diagnosis of PTSD, referral to PTSD specialist clinicians might be appropriate. It is possible that patients diagnosed with DS should routinely be screened for trauma history and post-traumatic symptomology.

#### **10.4. Directions for future research**

The current section includes suggestions for future research pertaining to the studies of emotional processing only. As mentioned previously, further research may seek to compare emotional processing in DS patients with additional clinical groups. For example, it might be valuable to include a group of participants reporting symptoms of anxiety or depression only (without any dissociative, conversion or somatoform disorder/symptoms), in order to provide a more rigorous control for these symptoms than can be achieved using statistical methods (as in the current study). On the other hand, a control group of patients with PTSD symptoms without DS could be included in future studies, in order to differentiate them from traumatised patients with DS (as carried out by Roberts et al., 2012, for example), and thus examine specifically what is unique about traumatised individuals who develop DS compared to individuals who develop more common post-traumatic symptoms (e.g. re-experiencing). Furthermore, in terms of establishing similarities or differences between DS and other dissociative/conversion disorders, an additional control group from one of these categories (e.g. conversion paralysis, conversion movement disorder, depersonalisation/derealisation disorder) might also be of interest.

The possibility of using additional experimental paradigms to examine emotional processing should also be considered. The paradigms utilised in the studies reported here are fairly well established techniques that have been used widely in other clinical groups. However, there are a variety of other possible techniques that could be used to extend the current findings, as several questions remain unanswered. Regarding preconscious processes, it is important that additional studies aim to provide further examination of the extent to which hypervigilance to emotional facial expressions occurs on a preconscious or conscious level. Other paradigms could be utilised that allow the manipulation of such stimuli and the measurement of subsequent behaviours, such as the dot probe paradigm (MacLeod, Mathews, & Tata, 1986) or

other priming paradigms. The degree of awareness of the stimuli could be systematically varied by altering presentation time, and it might be interesting to conduct between-groups analyses between patients who have different degrees of awareness. This would allow an examination of the possible influence of conscious awareness on emotional responding, for example.

Regarding explicit facial expression processing, it might be of value to explore the use of 'morphing' paradigms (e.g. Calder et al., 1996), in which participants are required to detect a specific emotion in facial expressions that systemically vary from neutral to the target emotional expression in small successive steps. This type of paradigm is more comparable to detecting dynamic changes in facial expressions in real-life than viewing static prototypical images of faces, and it is possible that this might be of relevance in this population (i.e. given the proposed hypervigilance to such signals).

A clear direction for future research is to further examine emotional processing of stimuli that extends beyond facial expressions. Given the fact that exaggerated autonomic responding to general affective stimuli was observed in Chapter 8, it would be of interest to examine whether this patient group show preconscious attentional biases towards such stimuli, or particular categories of them (e.g. negative stimuli). This would indicate whether the preconscious bias observed in Chapter 6 extended to other, more varied stimuli. Another possibility would be to study emotional processing of trauma-related stimuli in patients with DS. Whilst there are ethical concerns that would need to be addressed in carrying out such a study, the possible theoretical and practical value may justify the use of such methods.

It might also be relevant to explore whether there are attentional biases towards stimuli (words, pictures) that relate to physical trauma (e.g. injury, surgery, illness) or psychological trauma (interpersonal conflict, violence, abuse). If attentional biases to such stimuli were revealed, there would be direct and important theoretical and therapeutic implications. Studies in patients with PTSD and borderline personality disorder have utilised personalised trauma scripts, yielding important insights (e.g. Elzinga et al., 2003; Schmahl et al., 2004). This type of approach might allow

investigators to more precisely examine the specific types of traumatic experience of relevance to patients with DS.

Moreover, as seen in other psychological disorders, emotional processing biases often tend to relate to stimuli that are somehow directly relevant to the symptoms of the disorder itself (e.g. threat and anxiety, negative stimuli and depression, physical symptom stimuli in somatoform disorder, see Chapter 6, section 6.1). Therefore, an important next step in this area would be to include stimuli that have direct relevance to the symptoms of DS, in order to examine the extent to which patients exhibit particular cognitive biases that might underlie or contribute to the occurrence of their seizures. These could involve words linked to seizure symptoms (e.g. drop, faint, swoon, shake, blank, panic, attack, paralysed, fear). Given the wide variety of different presentations, such stimuli could be personalised for each patient included. Another possibility is the use of images relating to the seizures, which could either be generic or again, could be personalised for each patient. The 'Implicit Association Test' measures implicit attitudes and could be used to further examine patients' implicit cognitive schemas about their symptoms, for example. Recently, Dimaro et al. (2014) used a similar measure to examine anxiety-related beliefs in DS patients (see Chapter 3, section 3.5.2). Again, such studies might directly inform cognitive interventions in the disorder.

It seems that a crucial area of additional research is to further explore emotional processes leading up to, and around the time of individual seizures. This would allow further examination of some of the hypotheses generated by the qualitative studies and those proposed to trigger DS in the model presented in the current chapter (Figure 15). Such studies might aim to monitor sympathetic arousal or activation of the HPA-axis on an ongoing basis for a given period of time, perhaps taking measurements at regular intervals. These physiological measures might be combined with the use of experience sampling (e.g. Csikszentmihalyi & Larson, 1987) to examine subtle fluctuations in mood and emotion leading up to and following individual seizures. A pragmatic and potentially feasible way of conducting such a study might be during inpatient stays in an epilepsy monitoring unit. Such a study carried out in this setting would have the advantage that more factors could be controlled (e.g.



nutrition, activity, substance use) than if it were carried out on an outpatient basis. This type of study would also represent a prospective design, in which the research staff and patients would be blind to diagnosis, although the possibility of a DS diagnosis might be known.

Finally, in terms of research design, a longitudinal study of emotional processing in this group is likely to be particularly informative, perhaps with measurements taken at initial onset of the disorder and then at intervals over its course (possibly before and after treatments). This would provide further evidence as to the direction of causality between emotional processing biases and DS.

### **10.5. Conclusions**

To conclude, DS are complex and severely disruptive phenomena. The remote causes of the disorder are likely to be the combined presence of a variety of risk factors, most likely rooted in developmental trauma and/or severe/multiple stresses. These life experiences seem to cause or amplify specific alterations in psychological functioning (e.g. dissociative tendencies, affect dysregulation, avoidant coping, depression/anxiety), and may combine with other vulnerabilities (e.g. neurological differences, cognitive deficits) to increase the risk of the initial onset of DS. Shaping variables, such as exposure to symptom models (i.e. family member DS or ES) may serve to direct these vulnerabilities towards DS rather than a different type of MUS. In adulthood, adverse life events (e.g. medical trauma, general traumatic events, multiple stressors, relationship crises) and ongoing psychological vulnerability (e.g. elevated stress/arousal levels, dissociative tendencies, PTSD, depression/anxiety) may elevate emotional dysfunction to such a level that the individual's cognitive, affective and behavioural means of coping are no longer sufficient, and thus, affective arousal may reach an intolerable threshold. At this point, the initial presentation of DS is proposed to be precipitated and the first seizure occur.

Within this thesis, it has been argued that DS are acute and severe dissociative states, characterised by losses in voluntary control and awareness of cognitive, affective, sensorimotor and behavioural processes. This acute dissociative state has been proposed to account for the variety of symptoms observed in, and reported by

patients with DS. Furthermore, it has been suggested that this extreme manifestation of dissociation (DS), may be related to and possibly exacerbated by a more general 'trait' style of dissociative responding. DS as a dissociative state, are hypothesised to be triggered by an acute elevation in already raised levels of affective arousal. The generally elevated levels of affective arousal seem to be perpetuated by a variety of psychosocial factors that may differ between patients.

Importantly, the results presented in the thesis have provided additional evidence for aberrant processing of affective stimuli in patients with DS. These differences include a preconscious hypervigilance for emotional facial expressions, alongside reduced explicit recognition and autonomic responding to such stimuli when they are available to conscious awareness. Moreover, the findings have suggested a tendency towards exaggerated autonomic responsivity to more general affective visual images, in the absence of any alteration in subjective responding to these stimuli, in patients with DS.

Together, the findings indicate that these important differences in emotional processing could play a critical role in the psychological mechanism that underlies DS occurrence. Specifically, these aberrant emotional processes might contribute to acute elevations in affective distress and arousal which would serve to trigger a severe and disruptive dissociative reaction. Therefore, these aberrant affective processes could be seen as the proximal cause of the occurrence of individual DS episodes, in at least a subgroup of individuals with the diagnosis.

## **References**

- Aboukasm, A., Mahr, G., Gahry, B. R., Thomas, A., & Barkley, G. L. (1998). Retrospective analysis of the effects of psychotherapeutic interventions on outcomes of psychogenic nonepileptic seizures. *Epilepsia*, 39(5), 470-473.
- Abubakr, A., Kablinger, A., & Caldito, G. (2003). Psychogenic seizures: Clinical features and psychological analysis. *Epilepsy & Behavior*, 4(3), 241-245.
- Abubakr, A., & Wambacq, I. (2005). Seizures in the elderly: Video/EEG monitoring analysis. *Epilepsy & Behavior*, 7(3), 447-450. doi:10.1016/j.yebeh.2005.06.012
- Abubakr, A., & Wambacq, I. (2013). Psychogenic nonepileptic seizures in men: Comparison of clinical and psychosocial features among Afro-American and Caucasian patients. *Epilepsia*, 54, 106-107. doi:http://dx.doi.org/10.1111/epi.12229
- Abubakr, A., Wambacq, I., & Goerres, H. (2013). Psychogenic non-epileptic seizures: Comparison of clinical manifestations between Afro-American and Caucasian female patients. *Journal of Clinical Neurophysiology*, 30(3), 229. doi:http://dx.doi.org/10.1097/WNP.0b013e318290ffba
- ADInstruments. (2008). *GSR Amp: Owner's guide*. New South Wales: ADInstruments Pty Ltd.
- Ahmedani, B. K., Osborne, J., Nerenz, D. R., Haque, S., Pietrantonio, L., Mahone, D., & Smith, B. J. (2013). Diagnosis, costs, and utilization for psychogenic non-epileptic seizures in a US health care setting. *Psychosomatics*, 54(1), 28-34. doi:http://dx.doi.org/10.1016/j.psych.2012.08.005
- Akyuz, G., Kugu, N., Akyuz, A., & Dogan, O. (2004). Dissociation and childhood abuse history in epileptic and pseudoseizure patients. *Epileptic Disorders*, 6(3), 187-192.

Al Marzooqi, S. M., Baker, G. A., Reilly, J., & Salmon, P. (2004). The perceived health status of people with psychologically derived non-epileptic attack disorder and epilepsy: A comparative study. *Seizure*, 13(2), 71-75. doi:10.1016/S1059-1311(03)00158-4

Aldenkamp, A. P., Krom, M. D., & Reijls, R. (2003). Newer antiepileptic drugs and cognitive issues. *Epilepsia*, 44(s4), 21-29.

Alessi, R., & Valente, K. (2014). Antiepileptic drugs in patients with psychogenic nonepileptic seizures: Lack of response is not mandatory and may delay diagnosis. *Epilepsy Currents*, 14, 204.

Alessi, R., & Valente, K. (2013). Psychogenic non-epileptic seizures at a tertiary care center in Brazil. *Epilepsy and Behavior*, 26(1), 91-95.  
doi:http://dx.doi.org/10.1016/j.yebeh.2012.10.011

Alessi, R., Vincentiis, S., Rzezak, P., & Valente, K. (2013). Semiology of psychogenic nonepileptic seizures: Age-related differences. *Epilepsy and Behavior*, 27(2), 292-295.  
doi:http://dx.doi.org/10.1016/j.yebeh.2013.02.003

Ali, F., Rickards, H., Bagary, M., Greenhill, L., McCorry, D., & Cavanna, A. E. (2010). Ictal consciousness in epilepsy and nonepileptic attack disorder. *Epilepsy & Behavior*, 19(3), 522-525. doi:http://dx.doi.org/10.1016/j.yebeh.2010.08.014

Allendorfer, J. B., & Szaflarski, J. P. (2014). Contributions of fMRI towards our understanding of the response to psychosocial stress in epilepsy and psychogenic nonepileptic seizures. *Epilepsy and Behavior*, 35, 19-25.  
doi:http://dx.doi.org/10.1016/j.yebeh.2014.03.023

Almis, B. H., Cumurcu, B. E., Unal, S., Ozcan, A. C., & Aytas, O. (2013). The neuropsychological and neurophysiological profile of women with pseudoseizure. *Comprehensive Psychiatry*, 54(6), 649-657. doi:10.1016/j.comppsy.2012.12.027

Alper, K., Devinsky, O., Perrine, K., Luciano, D., Vazquez, B., Pacia, S., & Rhee, E. (1997). Dissociation in epilepsy and conversion nonepileptic seizures. *Epilepsia*, 38(9), 991-997. doi:10.1111/j.1528-1157.1997.tb01481.x

Alper, K., Devinsky, O., Perrine, K., Vazquez, B., & Luciano, D. (1993). Nonepileptic seizures and childhood sexual and physical abuse. *Neurology*, 43(10), 1950-1953.

Alper, K., Devinsky, O., Perrine, K., Vazquez, B., & Luciano, D. (1995). Psychiatric classification of nonconversion nonepileptic seizures. *Archives of Neurology*, 52(2), 199-201.

Alsaadi, T. M., & Marquez, A. V. (2005). Psychogenic nonepileptic seizures. *American Family Physician*, 72(5), 849-856.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders - fourth edition (DSM-IV)*. Washington, DC: American Psychiatric Association.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders – fifth edition. (DSM-5)*. Washington, DC: American Psychiatric Association.

An, D., Wu, X., Yan, B., Mu, J., & Zhou, D. (2010). Clinical features of psychogenic nonepileptic seizures: A study of 64 cases in southwest China. *Epilepsy and Behavior*, 17(3), 408-411. doi:http://dx.doi.org/10.1016/j.yebeh.2010.01.003

Anderson, G., Yassenik, L., & Ross, C. A. (1993). Dissociative experiences and disorders among women who identify themselves as sexual abuse survivors. *Child Abuse & Neglect*, 17(5), 677-686.

Andrade, C., Singh, N. M., & Bhakta, S. G. (2006). Simultaneous true seizures and pseudoseizures. *The Journal of Clinical Psychiatry*, 67(4), 673.

Arain, A. M., Hamadani, A. M., Islam, S., & Abou-Khalil, B. W. (2007). Predictors of early seizure remission after diagnosis of psychogenic nonepileptic seizures. *Epilepsy and Behavior*, 11(3), 409-412. doi:http://dx.doi.org/10.1016/j.yebeh.2007.07.017

- Arce, E., Simmons, A. N., Lovero, K. L., Stein, M. B., & Paulus, M. P. (2008). Escitalopram effects on insula and amygdala BOLD activation during emotional processing. *Psychopharmacology*, 196(4), 661-672.
- Arnold, L. M., & Privitera, M. D. (1996). Psychopathology and trauma in epileptic and psychogenic seizure patients. *Psychosomatics*, 37(5), 438-443.  
doi:[http://dx.doi.org/10.1016/S0033-3182\(96\)71531-1](http://dx.doi.org/10.1016/S0033-3182(96)71531-1)
- Arntz, A., Appels, C., & Sieswerda, S. (2000). Hypervigilance in borderline disorder: A test with the emotional Stroop paradigm. *Journal of Personality Disorders*, 14(4), 366-373.
- Arthuis, M., Micoulaud-Franchi, J. A., Bartolomei, F., McGonigal, A., & Guedj, E. (2014). Resting cortical PET metabolic changes in psychogenic non-epileptic seizures (PNES). *Journal of Neurology, Neurosurgery, and Psychiatry*, doi:jnnp-2014-309390 [pii]
- Arzy, S., Halje, P., Schechter, D. S., Spinelli, L., Seeck, M., & Blanke, O. (2014). Neural generators of psychogenic seizures: Evidence from intracranial and extracranial brain recordings. *Epilepsy & Behavior*, 31(0), 381-385.  
doi:<http://dx.doi.org/10.1016/j.yebeh.2013.10.017>
- Asadi-Pooya, A. A., & Emami, M. (2013). Demographic and clinical manifestations of psychogenic non-epileptic seizures: The impact of co-existing epilepsy in patients or their family members. *Epilepsy and Behavior*, 27(1), 1-3.  
doi:<http://dx.doi.org/10.1016/j.yebeh.2012.12.010>
- Asadi-Pooya, A. A., Emami, M., & Emami, Y. (2013). Gender differences in manifestations of psychogenic non-epileptic seizures in Iran. *Journal of the Neurological Sciences*, 332(1-2), 66-68. doi:<http://dx.doi.org/10.1016/j.jns.2013.06.016>
- Asadi-Pooya, A. A., Emami, Y., & Emami, M. (2014). Psychogenic non-epileptic seizures in Iran. *Seizure*, 23(3), 175-177. doi:<http://dx.doi.org/10.1016/j.seizure.2013.11.005>

Asadi-Pooya, A. A., Emami, Y., Emami, M., & Sperling, M. R. (2014). Prolonged psychogenic nonepileptic seizures or pseudostatus. *Epilepsy and Behavior*, 31, 304-306. doi:<http://dx.doi.org/10.1016/j.yebeh.2013.10.019>

Asmussen, S. B., Kirlin, K. A., Gale, S. D., & Chung, S. S. (2009). Differences in self-reported depressive symptoms between patients with epileptic and psychogenic nonepileptic seizures. *Seizure*, 18(8), 564-566. doi:10.1016/j.seizure.2009.05.006

Ataoglu, A., Ozcetin, A., Icmeli, C., & Ozbulut, O. (2003). Paradoxical therapy in conversion reaction. *Journal of Korean Medical Science*, 18(4), 581-584. doi:200308581 [pii]

Avbersek, A., & Sisodiya, S. (2010). Does the primary literature provide support for clinical signs used to distinguish psychogenic nonepileptic seizures from epileptic seizures? *Journal of Neurology, Neurosurgery and Psychiatry*, 81(7), 719-725. doi:<http://dx.doi.org/10.1136/jnnp.2009.197996>

Awad, H., & Softic, J. (2011). Psychical and psychological characteristics of patients with non-epileptic seizures. *Medicinski Glasnik*, 8(2), 224-228.

Azar, N. J., Pitiyanuvath, N., Vittal, N. B., Wang, L., Shi, Y., & Abou-Khalil, B. W. (2010). A structured questionnaire predicts if convulsions are epileptic or nonepileptic. *Epilepsy & Behavior*, 19(3), 462-466. doi:10.1016/j.yebeh.2010.08.027

Azar, N. J., Tayah, T. F., Wang, L., Song, Y., & Abou-Khalil, B. W. (2008). Postictal breathing pattern distinguishes epileptic from nonepileptic convulsive seizures. *Epilepsia*, 49(1), 132-137. doi:10.1111/j.1528-1167.2007.01215.x

Baer, R. A., Peters, J. R., Eisenlohr-Moul, T. A., Geiger, P. J., & Sauer, S. E. (2012). Emotion-related cognitive processes in borderline personality disorder: A review of the empirical literature. *Clinical Psychology Review*, 32(5), 359-369.

Bagby, R. M., Parker, J. D., & Taylor, G. J. (1994). The twenty-item Toronto Alexithymia Scale—I. item selection and cross-validation of the factor structure. *Journal of Psychosomatic Research*, 38(1), 23-32.

Baillés, E., Pintor, L., Fernandez-Egea, E., Torres, X., Matrai, S., de Pablo, J., & Arroyo, S. (2004). Psychiatric disorders, trauma, and MMPI profile in a Spanish sample of nonepileptic seizure patients. *General Hospital Psychiatry*, 26(4), 310-315.  
doi:10.1016/j.genhosppsych.2004.04.003

Baker, G. A., Brooks, J. L., Goodfellow, L., Bodde, N., & Aldenkamp, A. (2007). Treatments for non-epileptic attack disorder. *Cochrane Database of Systematic Reviews*, (1), CD006370. doi:10.1002/14651858.CD006370

Baker, G. A., Moore, P., & Appleton, R. E. (1995). Nonepileptic attack disorders in children and adolescents - a single-case study. *Seizure*, 4(4), 307-309.  
doi:10.1016/S1059-1311(95)80009-3

Bakvis, P., Roelofs, K., Kuyk, J., Edelbroek, P. M., Swinkels, W. A. M., & Spinhoven, P. (2009). Trauma, stress, and preconscious threat processing in patients with psychogenic nonepileptic seizures. *Epilepsia*, 50(5), 1001-1011. doi:10.1111/j.1528-1167.2008.01862.x

Bakvis, P., Spinhoven, P., Giltay, E. J., Kuyk, J., Edelbroek, P. M., Zitman, F. G., & Roelofs, K. (2010). Basal hypercortisolism and trauma in patients with psychogenic nonepileptic seizures. *Epilepsia*, 51(5), 752-759. doi:http://dx.doi.org/10.1111/j.1528-1167.2009.02394.x

Bakvis, P., Spinhoven, P., Putman, P., Zitman, F. G., & Roelofs, K. (2010). The effect of stress induction on working memory in patients with psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 19(3), 448-454. doi:10.1016/j.yebeh.2010.08.026



Bakvis, P., Spinhoven, P., & Roelofs, K. (2009). Basal cortisol is positively correlated to threat vigilance in patients with psychogenic nonepileptic seizures. *Epilepsy and Behavior*, 16(3), 558-560. doi:<http://dx.doi.org/10.1016/j.yebeh.2009.09.006>

Bakvis, P., Spinhoven, P., Zitman, F. G., & Roelofs, K. (2011). Automatic avoidance tendencies in patients with psychogenic non epileptic seizures. *Seizure-European Journal of Epilepsy*, 20(8), 628-634. doi:10.1016/j.seizure.2011.06.006

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & Van Ijzendoorn, M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: A meta-analytic study. *Psychological Bulletin*, 133(1), 1.

Barnes-Holmes, D., Barnes-Holmes, Y., Power, P., Hayden, E., Milne, R., & Stewart, I. (2006). Do you really know what you believe? Developing the implicit relational assessment procedure (IRAP) as a direct measure of implicit beliefs. *The Irish Psychologist*, 32(7), 169-177.

Barry, E., Krumholz, A., Bergey, G. K., Chatha, H., Alemayehu, S., & Grattan, L. (1998). Nonepileptic posttraumatic seizures. *Epilepsia*, 39(4), 427-431. doi:10.1111/j.1528-1157.1998.tb01395.x

Barry, J. J., Atzman, O., & Morrell, M. J. (2000). Discriminating between epileptic and nonepileptic events: The utility of hypnotic seizure induction. *Epilepsia*, 41(1), 81-84.

Barry, J. J., Wittenberg, D., Bullock, K. D., Michaels, J. B., Classen, C. C., & Fisher, R. S. (2008). Group therapy for patients with psychogenic nonepileptic seizures: A pilot study. *Epilepsy & Behavior*, 13(4), 624-629.  
doi:<http://dx.doi.org/10.1016/j.yebeh.2008.06.013>

Baslet, G. (2011). Psychogenic non-epileptic seizures: A model of their pathogenic mechanism. *Seizure*, 20(1), 1-13. doi:<http://dx.doi.org/10.1016/j.seizure.2010.10.032>

- Baslet, G. (2012). Psychogenic nonepileptic seizures: A treatment review. What have we learned since the beginning of the millennium? *Neuropsychiatric Disease and Treatment*, 8, 585-598. doi:<http://dx.doi.org/10.2147/NDT.S32301>
- Baslet, G., Dworetzky, B., Perez, D. L., & Oser, M. (2014). Treatment of psychogenic nonepileptic seizures: Updated review and findings from a mindfulness-based intervention case series. *Clinical EEG and Neuroscience*, doi:1550059414557025 [pii]
- Baslet, G., & Hill, J. (2011). Case report: Brief mindfulness-based psychotherapeutic intervention during inpatient hospitalization in a patient with conversion and dissociation. *Clinical Case Studies*, 10 (2), 95-109, doi: 10.1177/1534650110396359
- Baslet, G., Roiko, A., & Prensky, E. (2010). Heterogeneity in psychogenic nonepileptic seizures: Understanding the role of psychiatric and neurological factors. *Epilepsy & Behavior*, 17(2), 236-241. doi:<http://dx.doi.org/10.1016/j.yebeh.2009.12.008>
- Bateman, A. W., & Fonagy, P. (2004). Mentalization-based treatment of BPD. *Journal of Personality Disorders*, 18(1), 36-51.
- Bateman, A.W., & Fonagy, P. (2006). *Mentalization-based treatment for borderline personality disorder: A practical guide*. Oxford: Oxford University Press.
- Bauer, J., & Elger, C. E. (2001). Psychogenic seizures mimicking juvenile myoclonic epilepsy: Case reports. *Seizure*, 10(3), 208-211. doi:<http://dx.doi.org/10.1053/seiz.2000.0488>
- Bautista, R. E., Gonzales-Salazar, W., & Ochoa, J. G. (2008). Expanding the theory of symptom modeling in patients with psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 13(2), 407-409. doi:10.1016/j.yebeh.2008.04.016.
- Baxter, S., Mayor, R., Baird, W., Brown, R., Cock, H., Howlett, S., . . . Reuber, M. (2012). Understanding patient perceptions following a psycho-educational intervention for psychogenic non-epileptic seizures. *Epilepsy & Behavior*, 23(4), 487-493. doi:10.1016/j.yebeh.2011.12.002

Bazil, C. W., Kothari, M., Luciano, D., Moroney, J., Song, S., Vasquez, B., . . . Devinsky, O. (1994). Provocation of nonepileptic seizures by suggestion in a general seizure population. *Epilepsia*, 35(4), 768-770. doi:10.1111/j.1528-1157.1994.tb02509.x

Bazil, C. W., & Walczak, T. S. (1997). Effects of sleep and sleep stage on epileptic and nonepileptic seizures. *Epilepsia*, 38(1), 56-62. doi:http://dx.doi.org/10.1111/j.1528-1157.1997.tb01077.x

Behrouz, R., Heriaud, L., & Benbadis, S. (2004). Late-onset psychogenic nonepileptic seizures. *Epilepsia*, 45, 256-256.

Bell, V., Oakley, D. A., Halligan, P. W., & Deeley, Q. (2011). Dissociation in hysteria and hypnosis: Evidence from cognitive neuroscience. *Journal of Neurology, Neurosurgery, and Psychiatry*, 82(3), 332-339. doi:10.1136/jnnp.2009.199158 [doi]

Bell, W. L., Park, Y. D., Thompson, E. A., & Radtke, R. A. (1998). Ictal cognitive assessment of partial seizures and pseudoseizures. *Archives of Neurology*, 55(11), 1456-1459.

Benbadis, S. R. (1999). How many patients with pseudoseizures receive antiepileptic drugs prior to diagnosis? *European Neurology*, 41(2), 114-115. doi:10.1159/000008015

Benbadis, S. R. (2005a). A spell in the epilepsy clinic and a history of "chronic pain" or "fibromyalgia" independently predict a diagnosis of psychogenic seizures. *Epilepsy & Behavior*, 6(2), 264-265. doi:10.1016/j.yebeh.2004.12.007

Benbadis, S. R. (2005b). The problem of psychogenic symptoms: Is the psychiatric community in denial? *Epilepsy & Behavior*, 6(1), 9-14. doi:10.1016/j.yebeh.2004.10.009

Benbadis, S.R. (2009). The differential diagnosis of epilepsy: A critical review. *Epilepsy & Behavior*, 15(1), 15-21. doi:http://dx.doi.org/10.1016/j.yebeh.2009.02.024

Benbadis, S. R. (2010). Psychogenic nonepileptic "seizures" or "attacks"? It's not just semantics: Attacks. *Neurology*, 75(1), 84-86. doi:10.1212/WNL.0b013e3181e6216f; 10.1212/WNL.0b013e3181e6216f

Benbadis, S. R., Agrawal, V., & Tatum W. O. (2001). How many patients with psychogenic nonepileptic seizures also have epilepsy? *Neurology*, 57(5), 915-917.

Benbadis, S. R., & Allen Hauser, W. (2000). An estimate of the prevalence of psychogenic non-epileptic seizures. *Seizure*, 9(4), 280-281. doi:10.1053/seiz.2000.0409

Benbadis, S. R., Blustein, J. N., & Sunstad, L. (2000). Should patients with psychogenic nonepileptic seizures be allowed to drive? *Epilepsia*, 41(7), 895-897.

Benbadis, S. R., Johnson, K., Anthony, K., Caines, G., Hess, G., Jackson, C., . . . Tatum, W. O. (2000). Induction of psychogenic nonepileptic seizures without placebo. *Neurology*, 55(12), 1904-1905.

Benbadis, S. R., & LaFrance Jr, W. C. (2010). Clinical features and the role of video-EEG monitoring. In S. C. Schachter, & W. C. LaFrance Jr (Eds.), *Gates and Rowan's nonepileptic seizures* (pp. 38-50). Cambridge, UK: Cambridge University Press.

Benbadis, S. R., Siegrist, K., Tatum, W. O., Heriaud, L., & Anthony, K. (2004). Short-term outpatient EEG video with induction in the diagnosis of psychogenic seizures. *Neurology*, 63(9), 1728-1730.

Benbadis, S. R., Tatum W. O., Murtagh, F. R., & Vale, F. L. (2000). MRI evidence of mesial temporal sclerosis in patients with psychogenic nonepileptic seizures. *Neurology*, 55(7), 1061-1062.

Benton, A. L., Sivan, A. B., Hamsher, K. D., Varney, N. R., & Spreen, O. (1994). *Contributions to neuropsychological assessment: A clinical manual* (2nd ed.). Oxford: Oxford University Press.

- Berkhoff, M., Briellmann, R. S., Radanov, B. P., Donati, F., & Hess, C. W. (1998). Developmental background and outcome in patients with nonepileptic versus epileptic seizures: A controlled study. *Epilepsia*, 39(5), 463-469.
- Bernstein, E. M., & Putnam, F. W. (1986). Development, reliability, and validity of a dissociation scale. *The Journal of Nervous and Mental Disease*, 174(12), 727-735.
- Betts, T., & Boden, S. (1992a). Diagnosis, management and prognosis of a group of 128 patients with non-epileptic attack disorder. Part I. *Seizure*, 1(1), 19-26.
- Betts, T., & Boden, S. (1992b). Diagnosis, management and prognosis of a group of 128 patients with non-epileptic attack disorder. Part II. Previous childhood sexual abuse in the aetiology of these disorders. *Seizure*, 1(1), 27-32.
- Bewley, J., Murphy, P. N., Mallows, J., & Baker, G. A. (2005). Does alexithymia differentiate between patients with nonepileptic seizures, patients with epilepsy, and nonpatient controls? *Epilepsy & Behavior*, 7(3), 430-437. doi:10.1016/j.yebeh.2005.06.006
- Bhatia, M. S., & Sapra, S. (2005). Pseudoseizures in children: A profile of 50 cases. *Clinical Pediatrics*, 44(7), 617-621.
- Bhatia, M., Sinha, P. K., Jain, S., Padma, M. V., & Maheshwari, M. C. (1997). Usefulness of short-term video EEG recording with saline induction in pseudoseizures. *Acta Neurologica Scandinavica*, 95(6), 363-366.
- Biehl, M., Matsumoto, D., Ekman, P., Hearn, V., Heider, K., Kudoh, T., & Ton, V. (1997). Matsumoto and Ekman's Japanese and Caucasian facial expressions of emotion (JACFEE): Reliability data and cross-national differences. *Journal of Nonverbal Behavior*, 21(1), 3-21.
- Binder, L. M., & Campbell, K. A. (2004). Medically unexplained symptoms and neuropsychological assessment. *Journal of Clinical and Experimental Neuropsychology*, 26(3), 369-392.

- Binder, L. M., Kindermann, S. S., Heaton, R. K., & Salinsky, M. C. (1998). Neuropsychologic impairment in patients with nonepileptic seizures. *Archives of Clinical Neuropsychology*, 13(6), 513-522. doi:<http://dx.doi.org/10.1016/S0887-6177%2897%2900042-5>
- Binder, L. M., & Salinsky, M. C. (2007). Psychogenic nonepileptic seizures. *Neuropsychology Review*, 17(Special Issue Neuropsychology of Epilepsy: Current Trends.), 405-412. doi:<http://dx.doi.org/10.1007/s11065-007-9047-5>
- Binder, L. M., Storzbach, D., Campbell, K. A., Rohlman, D. S., Anger, W. K., Salinsky, M. C., . . . Mueller, R. (2000). Comparison of MMPI-2 profiles of Gulf war veterans with epileptic and nonepileptic seizure patients. *Assessment*, 7(1), 73-78.
- Binzer, M., Stone, J., & Sharpe, M. (2004). Recent onset pseudoseizures - clues to aetiology. *Seizure*, 13(3), 146-155. doi:10.1016/S1059-1311(03)00184-5.
- Bisson, J. I., Ehlers, A., Matthews, R., Pilling, S., Richards, D., & Turner, S. (2007). Psychological treatments for chronic post-traumatic stress disorder. Systematic review and meta-analysis. *The British Journal of Psychiatry*, 190, 97-104. doi:190/2/97 [pii]
- Black, D. N., Seritan, A. L., Taber, K. H., & Hurley, R. A. (2004). Conversion hysteria: Lessons from functional imaging. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 16(3), 245-251. doi:10.1176/appi.neuropsych.16.3.245 [doi]
- Black, L. C., Schefft, B. K., Howe, S. R., Szaflarski, J. P., Yeh, H., & Privitera, M. D. (2010). The effect of seizures on working memory and executive functioning performance. *Epilepsy & Behavior*, 17(3), 412-419. doi:10.1016/j.yebeh.2010.01.006
- Bland, A. R., Williams, C. A., Scharer, K., & Manning, S. (2004). Emotion processing in borderline personality disorders. *Issues in Mental Health Nursing*, 25(7), 655-672.
- Blumer, D., Rice, S., & Adamolekun, B. (2009). Electroconvulsive treatment for nonepileptic seizure disorders. *Epilepsy & Behavior*, 15(3), 382-387. doi:<http://dx.doi.org/10.1016/j.yebeh.2009.05.004>

Bodde, N. M. G., Bartelet, D. C. J., Ploegmakers, M., Lazeron, R. H. C., Aldenkamp, A. P., & Boon, P. A. J. M. (2011). MMPI-II personality profiles of patients with psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 20(4), 674-680.

doi:<http://dx.doi.org/10.1016/j.yebeh.2011.01.034>

Bodde, N. M. G., Brooks, J. L., Baker, G. A., Boon, P. A. J. M., Hendriksen, J. G. M., Mulder, O. G., & Aldenkamp, A. P. (2009). Psychogenic non-epileptic seizures- definition, etiology, treatment and prognostic issues: A critical review. *Seizure*, 18(8), 543-553. doi:10.1016/j.seizure.2009.06.006

Bodde, N. M. G., Janssen, A. M. A. J., Theuns, C., Vanhoutvin, J. F. G., Boon, P. A. J. M., & Aldenkamp, A. P. (2007). Factors involved in the long-term prognosis of psychogenic nonepileptic seizures. *Journal of Psychosomatic Research*, 62(5), 545-551.

doi:10.1016/j.jpsychores.2006.11.015

Bodde, N. M. G., Lazeron, R. H. C., Wirken, J. M. A., van der Kruijs, S. J., Aldenkamp, A. P., & Boon, P. A. J. M. (2012). Patients with psychogenic non-epileptic seizures referred to a tertiary epilepsy centre: Patient characteristics in relation to diagnostic delay. *Clinical Neurology and Neurosurgery*, 114(3), 217-222.

doi:<http://dx.doi.org/10.1016/j.clineuro.2011.10.019>

Bodde, N.M., van der Kruijs, S., Ijff, D., Lazeron, R., Vonck, K., Boon, P., & Aldenkamp, A. (2013). Subgroup classification in patients with psychogenic non-epileptic seizures. *Epilepsy & Behavior*, 26(3), 279-289.

Boon, P. A., & Williamson, P. D. (1993). The diagnosis of pseudoseizures. *Clinical Neurology and Neurosurgery*, 95(1), 1-8.

Bora, I. H., Taskapilioglu, O., Seferoglu, M., Kotan, O. V., Bican, A., Ozkaya, G., & Akkaya, C. (2011). Sociodemographics, clinical features, and psychiatric comorbidities of patients with psychogenic nonepileptic seizures: Experience at a specialized epilepsy center in Turkey. *Seizure*, 20(6), 458-461.

doi:<http://dx.doi.org/10.1016/j.seizure.2011.02.007>

- Bortz, J. J., Prigatano, G. P., Blum, D., & Fisher, R. S. (1995). Differential response characteristics in nonepileptic and epileptic seizure patients on a test of verbal-learning and memory. *Neurology*, 45(11), 2029-2034.
- Boucsein, W., Fowles, D. C., Grimnes, S., Ben-Shakhar, G., Roth, W. T., Dawson, M. E., & Filion, D. L. (2012). Publication recommendations for electrodermal measurements. *Psychophysiology*, 49(8), 1017-1034.
- Bowman, E. S. (1993). Etiology and clinical course of pseudoseizures - relationship to trauma, depression, and dissociation. *Psychosomatics*, 34(4), 333-342.
- Bowman, E. S. (1998). Pseudoseizures. *Psychiatric Clinics of North America*, 21(3), 649-657. doi:10.1016/S0193-953X(05)70029-0
- Bowman, E. S. (2010). Posttraumatic stress disorder, abuse, and trauma: Relationships to psychogenic nonepileptic seizures. In S. C. Schachter, & W. C. LaFrance Jr (Eds.), *Gates and Rowan's nonepileptic seizures* (3rd ed., pp. 213-224). Cambridge: Cambridge University Press.
- Bowman, E. S., & Coons, P. M. (2000). The differential diagnosis of epilepsy, pseudoseizures, dissociative identity disorder, and dissociative disorder not otherwise specified. *Bulletin of the Menninger Clinic*, 64(2), 164-180.
- Bowman, E. S., & Markand, O. N. (1996). Psychodynamics and psychiatric diagnoses of pseudoseizure subjects. *American Journal of Psychiatry*, 153(1), 57-63.
- Bowman, E. S., & Markand, O. N. (1999). The contribution of life events to pseudoseizure occurrence in adults. *Bulletin of the Menninger Clinic*, 63(1), 70-88.
- Boysan, M., Goldsmith, R. E., Çavuş, H., Kayri, M., & Keskin, S. (2009). Relations among anxiety, depression, and dissociative symptoms: The influence of abuse subtype. *Journal of Trauma & Dissociation*, 10(1), 83-101.



- Bradley, B. P., Mogg, K., White, J., Groom, C., & Bono, J. (1999). Attentional bias for emotional faces in generalized anxiety disorder. *British Journal of Clinical Psychology*, 38(3), 267-278.
- Bradley, M. M., Codispoti, M., Cuthbert, B. N., & Lang, P. J. (2001). Emotion and motivation I: Defensive and appetitive reactions in picture processing. *Emotion*, 1(3), 276.
- Brady Jr., W. J., & Huff, J. S. (1997). Pseudotoxemia: New onset psychogenic seizure in third trimester pregnancy. *The Journal of Emergency Medicine*, 15(6), 815-820.  
doi:[http://dx.doi.org/10.1016/S0736-4679\(97\)00189-3](http://dx.doi.org/10.1016/S0736-4679(97)00189-3)
- Breh, D. C., & Seidler, G. H. (2007). Is peritraumatic dissociation a risk factor for PTSD? *Journal of Trauma & Dissociation*, 8(1), 53-69. doi:10.1300/J229v08n01\_04 [doi]
- Breier, J. I., Fuchs, K. L., Brookshire, B. L., Wheless, J., Thomas, A. B., Constantinou, J., & Willmore, L. J. (1998). Quality of life perception in patients with intractable epilepsy or pseudoseizures. *Archives of Neurology*, 55(5), 660-665.
- Bremner, J. D., Krystal, J. H., Putnam, F. W., Southwick, S. M., Marmar, C., Charney, D. S., & Mazure, C. M. (1998). Measurement of dissociative states with the clinician-administered dissociative states scale (CADSS). *Journal of Traumatic Stress*, 11(1), 125-136.
- Briere, J. (2000). *Inventory of altered self-capacities (IASC): Professional manual*. Odessa, FL: Psychological Assessment Resources.
- Briere, J. (2002). *Multiscale dissociation inventory (MDI): Professional manual*. Odessa: Psychological Assessment Resources.
- Briere, J. (2006). Dissociative symptoms and trauma exposure: Specificity, affect dysregulation, and posttraumatic stress. *The Journal of Nervous and Mental Disease*, 194(2), 78-82. doi:10.1097/01.nmd.0000198139.47371.54

Briere, J., Elliott, D. M., Harris, K., & Cotman, A. (1995). Trauma symptom inventory psychometrics and association with childhood and adult victimization in clinical samples. *Journal of Interpersonal Violence, 10*(4), 387-401.

Brooks, J. L., Goodfellow, L., Bodde, N. M. G., Aldenkamp, A., & Baker, G. A. (2007). Nondrug treatments for psychogenic nonepileptic seizures: What's the evidence? *Epilepsy and Behavior, 11*(3), 367-377. doi:<http://dx.doi.org/10.1016/j.yebeh.2007.05.007>

Brown, M. C., Levin, B. E., Ramsay, R. E., Katz, D. A., & Duchowny, M. S. (1991). Characteristics of patients with nonepileptic seizures. *Journal of Epilepsy, 4*(4), 225-229. doi:10.1016/0896-6974(91)90080-3

Brown, P., & van der Hart, O. (1998). Memories of sexual abuse: Janet's critique of Freud, a balanced approach. *Psychological Reports, 82*(3, Pt 1), 1027-1043. doi:10.2466/pr0.1998.82.3.1027

Brown, R. J. (2002). Epilepsy, dissociation and nonepileptic seizures. In M. R. Trimble, & B. Schmitz (Eds.), *The neuropsychiatry of epilepsy* (pp.189-209). Cambridge: Cambridge University Press.

Brown, R. J. (2004). Psychological mechanisms of medically unexplained symptoms: An integrative conceptual model. *Psychological Bulletin, 130*(5), 793.

Brown, R. J. (2006). Medically unexplained symptoms: A new model. *Psychiatry, 5*(2), 43-47.

Brown, R. J., Bouska, J. F., Frow, A., Kirkby, A., Baker, G. A., Kemp, S., . . . Reuber, M. (2013). Emotional dysregulation, alexithymia, and attachment in psychogenic nonepileptic seizures. *Epilepsy & Behavior, 29*(1), 178-183. doi:10.1016/j.yebeh.2013.07.019

Brown, R. J., Cardena, E., Nijenhuis, E., Sar, V., & van der Hart, O. (2007). Should conversion disorder be reclassified as a dissociative disorder in DSM-V? *Psychosomatics, 48*(5), 369-378. doi:10.1176/appi.psy.48.5.369

- Brown, R. J., Syed, T. U., Benbadis, S., LaFrance, W. C., & Reuber, M. (2011). Psychogenic nonepileptic seizures. *Epilepsy and Behavior*, 22(1), 85-93.  
doi:<http://dx.doi.org/10.1016/j.yebeh.2011.02.016>
- Brown, R. J., & Trimble, M. R. (2000). Dissociative psychopathology, non-epileptic seizures, and neurology. *Journal of Neurology Neurosurgery and Psychiatry*, 69(3), 285-288.  
doi:10.1136/jnnp.69.3.285
- Browning, M., Reid, C., Cowen, P. J., Goodwin, G. M., & Harmer, C. J. (2007). A single dose of citalopram increases fear recognition in healthy subjects. *Journal of Psychopharmacology*, 21(7), 684-690. doi:0269881106074062 [pii]
- Buchanan, N., & Snars, J. (1993). Pseudoseizures (non epileptic attack disorder) - clinical management and outcome in 50 patients. *Seizure*, 2(2), 141-146.  
doi:[http://dx.doi.org/10.1016/S1059-1311\(05\)80119-0](http://dx.doi.org/10.1016/S1059-1311(05)80119-0)
- Buckley, T. C., Blanchard, E. B., & Neill, W. T. (2000). Information processing and PTSD: A review of the empirical literature. *Clinical Psychology Review*, 20(8), 1041-1065.
- Buhler, K. E., & Heim, G. (2009). Psychopathological approaches in Pierre Janet's conception of the subconscious. *Psychopathology*, 42(3), 190-200.  
doi:10.1159/000209332 [doi]
- Burneo, J. G., Martin, R., Powell, T., Greenlee, S., Knowlton, R. C., Faught, R. E., . . . Kuzniecky, R. I. (2003). Teddy bears: An observational finding in patients with non-epileptic events. *Neurology*, 61(5), 714-715.
- Burnstine, T. H., Lesser, R. P., & Cole, A. J. (1991). Pseudoepileptiform EEG patterns during pseudoseizures. *Journal of Epilepsy*, 4(3), 165-171.  
doi:[http://dx.doi.org/10.1016/S0896-6974\(05\)80042-8](http://dx.doi.org/10.1016/S0896-6974(05)80042-8)
- Calder, A. J., Young, A. W., Perrett, D. I., Etcoff, N. L., & Rowland, D. (1996). Categorical perception of morphed facial expressions. *Visual Cognition*, 3(2), 81-118.

Cardeña, E. (1994). The domain of dissociation. In S. J. Lynn, & J. W. Rhue (Eds.), *Dissociation: Clinical and theoretical perspectives* (pp. 15-31). New York: Guildford Press.

Carlson Jr., R. H., & Caplan, J. P. (2011). Pseudolabor born out of psychogenic nonepileptic seizures: A case report of multisymptom conversion disorder. *Psychosomatics*, 52(5), 455-458. doi:<http://dx.doi.org/10.1016/j.psym.2011.01.017>

Carmant, L., Kramer, U., Mikati, M. A., Riviello, J. J., Helmers, S. L., & Holmes, G. L. (1995). Pseudoseizure manifestations in 2 preschool age children. *Seizure*, 4(2), 147-149. doi:10.1016/S1059-1311(95)80096-4

Carson, A. J., Brown, R., David, A. S., Duncan, R., Edwards, M. J., Goldstein, L. H., . . . UK-FNS. (2012). Functional (conversion) neurological symptoms: Research since the millennium. *Journal of Neurology Neurosurgery and Psychiatry*, 83(8), 842-850. doi:10.1136/jnnp-2011-301860

Carton, S., Thompson, P. J., & Duncan, J. S. (2003). Non-epileptic seizures: Patients' understanding and reaction to the diagnosis and impact on outcome. *Seizure*, 12(5), 287-294. doi:10.1016/S1059-1311(02)00290-X

Cervenka, M. C., Lesser, R., Tran, T. T., Fortune, T., Muthugovindan, D., & Miglioretti, D. L. (2013). Does the teddy bear sign predict psychogenic nonepileptic seizures? *Epilepsy and Behavior*, 28(2), 217-220. doi:<http://dx.doi.org/10.1016/j.yebeh.2013.05.016>

Chabolla, D. R., & Shih, J. J. (2006). Postictal behaviors associated with psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 9(2), 307-311. doi:10.1016/j.yebeh.2006.06.009

Chapleau, K. M., Landsberger, S. A., Povlinski, J., & Diaz, D. R. (2013). Using paradoxical intention therapy to treat refractory nonepileptic events. *Psychosomatics*, 54(4), 398-401. doi:<http://dx.doi.org/10.1016/j.psym.2012.12.003>

Chemali, Z., & Meadows, M. E. (2004). The use of eye movement desensitization and reprocessing in the treatment of psychogenic seizures. *Epilepsy & Behavior*, 5(5), 784-787. doi:10.1016/j.yebeh.2004.06.003

Chen, D. K., & Izadyar, S. (2010). Characteristics of psychogenic nonepileptic events among veterans with posttraumatic stress disorder: An association of semiology with the nature of trauma. *Epilepsy & Behavior*, 17(2), 188-192.  
doi:10.1016/j.yebeh.2009.11.012

Chen, D. K., Izadyar, S., Collins, R. L., Benge, J. F., LeMaire, A. W., & Hrachovy, R. A. (2011). Induction of psychogenic nonepileptic events: Success rate influenced by prior induction exposure, ictal semiology, and psychological profiles. *Epilepsia*, 52(6), 1063-1070. doi:10.1111/j.1528-1167.2011.02985.x

Chu, J. A. (1996). Posttraumatic responses to childhood abuse and implications for treatment. In L. K. Michelson, & W. J. Ray (Eds.), *Handbook of dissociation: Theoretical, empirical, and clinical perspectives* (pp. 381-397). New York: Plenum Press.

Chu, J. A., & Dill, D. L. (1990). Dissociative symptoms in relation to childhood physical and sexual abuse. *The American Journal of Psychiatry*, 147(7), 887-892.

Cianci, V., Ferlazzo, E., Condino, F., Mauvais, H. S., Farnarier, G., Labate, A., . . . Aguglia, U. (2011). Rating scale for psychogenic nonepileptic seizures: Scale development and clinimetric testing. *Epilepsy and Behavior*, 21(2), 128-131.  
doi:http://dx.doi.org/10.1016/j.yebeh.2011.03.025

Conwill, M., Oakley, L., Evans, K., & Cavanna, A. E. (2014). CBT-based group therapy intervention for nonepileptic attacks and other functional neurological symptoms: A pilot study. *Epilepsy & Behavior*, 34(0), 68-72.  
doi:http://dx.doi.org/10.1016/j.yebeh.2014.03.012

Cornaggia, C. M., Gugliotta, S. C., Magaudo, A., Alfa, R., Beghi, M., & Polita, M. (2012). Conversation analysis in the differential diagnosis of Italian patients with epileptic or

psychogenic non-epileptic seizures: A blind prospective study. *Epilepsy and Behavior*, 25(4), 598-604. doi:<http://dx.doi.org/10.1016/j.yebeh.2012.09.003>

Cowan, R. B. (2010). Psychogenic nonepileptic "seizures" or "attacks"? It's not just semantics: Seizures. *Neurology*, 75(23), 2135-6; author reply 2136-7.

Cragar, D. E., Berry, D. T., Fakhoury, T. A., Cibula, J. E., & Schmitt, F. A. (2002). A review of diagnostic techniques in the differential diagnosis of epileptic and nonepileptic seizures. *Neuropsychology Review*, 12(1), 31-64.

Cragar, D. E., Berry, D. T., Fakhoury, T. A., Cibula, J. E., & Schmitt, F. A. (2006). Performance of patients with epilepsy or psychogenic non-epileptic seizures on four measures of effort. *The Clinical Neuropsychologist*, 20(3), 552-566.  
doi:10.1080/13854040590947380

Cragar, D. E., Fakhoury, T. A., Berry, D. T. R., & Schmitt, F. A. (2004). Cluster analysis of normal personality traits in patients with psychogenic nonepileptic seizures. *Epilepsia*, 45, 172-173.

Crick, F., & Koch, C.A. (2007). A neurobiological framework for consciousness. In M.Velmans, & S. Schneider (Eds.), *The Blackwell Companion to Consciousness* (pp. 567-579). Oxford: Blackwell Publishing Ltd.

Cronje, G., & Pretorius, C. (2013). The coping styles and health-related quality of life of South African patients with psychogenic nonepileptic seizures. *Epilepsy and Behavior*, 29(3), 581-584. doi:<http://dx.doi.org/10.1016/j.yebeh.2013.09.045>

Csikszentmihalyi, M., & Larson, R. (1987). Validity and reliability of the experience-sampling method. *The Journal of Nervous and Mental Disease*, 175(9), 526-536.

D'Alessio, L., Giagante, B., Oddo, S., Silva W, W., Solís, P., Consalvo, D., & Kochen, S. (2006). Psychiatric disorders in patients with psychogenic non-epileptic seizures, with and without comorbid epilepsy. *Seizure*, 15(5), 333-339.  
doi:<http://dx.doi.org/10.1016/j.seizure.2006.04.003>

Dalenberg, C. J., Brand, B. L., Gleaves, D. H., Dorahy, M. J., Loewenstein, R. J., Cardeña, E., . . . Spiegel, D. (2012). Evaluation of the evidence for the trauma and fantasy models of dissociation. *Psychological Bulletin*, 138(3), 550-588.

Daros, A., Zakzanis, K., & Ruocco, A. (2013). Facial emotion recognition in borderline personality disorder. *Psychological Medicine*, 43(9), 1953-1963.

Darwin, C. R. (1872). *The expression of the emotions in man and animals*. London: Murray. Retrieved from: <http://darwin-online.org.uk/content/frameset?pageseq=1&itemID=F1142&viewtype=text>

Davies, K. G., Blumer, D. P., Lobo, S., Hermann, B. P., Phillips, B. L. B., & Montouris, G. D. (2000). De novo nonepileptic seizures after cranial surgery for epilepsy: Incidence and risk factors. *Epilepsy & Behavior*, 1(6), 436-443.  
doi:<http://dx.doi.org/10.1006/ebeh.2000.0124>

Davis, B. J. (2004). Predicting nonepileptic seizures utilizing seizure frequency, EEG, and response to medication. *European Neurology*, 51(3), 153-156. doi:10.1159/000077287

Dawson, M. E., Schell, A. M., & Fillion, D. L. (2000). The electrodermal system. In J. T. Cacioppo, L. G. Tassinary, & G. G. Bernston (Eds.), *Handbook of psychophysiology* (2nd ed., pp. 200-223). Cambridge: Cambridge University Press.

De Marco, F. A., Ghizoni, E., Kobayashi, E., Li, L. M., & Cendes, F. (2003). Cerebellar volume and long-term use of phenytoin. *Seizure*, 12(5), 312-315.

De Waal, M. W., Arnold, I. A., Eekhof, J. A., & van Hemert, A. M. (2004). Somatoform disorders in general practice: Prevalence, functional impairment and comorbidity with anxiety and depressive disorders. *The British Journal of Psychiatry*, 184, 470-476.

De Wet, C. J., Mellers, J. D.C., Gardner, W. N., & Toone, B. K. (2003). Pseudoseizures and asthma. *Journal of Neurology, Neurosurgery, and Psychiatry*, 74(5), 639-641.

Dehaene, S., Changeux, J., Naccache, L., Sackur, J., & Sergent, C. (2006). Conscious, preconscious, and subliminal processing: A testable taxonomy. *Trends in Cognitive Sciences*, 10(5), 204-211.

DeLeon, I. G., Uy, M., & Gutshall, K. (2005). Noncontingent reinforcement and competing stimuli in the treatment of pseudoseizures and destructive behaviors. *Behavioral Interventions*, 20(3), 203-217. doi:10.1002/bin.192

Dericioglu, N., Saygi, S., & Ciger, A. (1999). The value of provocation methods in patients suspected of having non-epileptic seizures. *Seizure*, 8(3), 152-156. doi:10.1053/seiz.1999.0277

Derogatis, L.R. (1993). *Brief symptom inventory (BSI)*. US: Pearson Clinical.

Derogatis, L. R. (1994). *Symptom checklist-90-revised (SCL-90-R)*. US: Pearson Clinical.

Derry, P. A., & McLachlan, R. S. (1996). The MMPI-2 as an adjunct to the diagnosis of pseudoseizures. *Seizure*, 5(1), 35-40. doi:http://dx.doi.org/10.1016/S1059-1311(96)80060-4

Desai, B. T., Porter, R. J., & Penry, J. K. (1982). Psychogenic seizures. A study of 42 attacks in six patients, with intensive monitoring. *Archives of Neurology*, 39(4), 202-209.

Desai, P., & Talwar, D. (1992). Nonepileptic events in normal and neurologically handicapped children: A video-EEG study. *Pediatric Neurology*, 8(2), 127-129. doi:0887-8994(92)90033-U [pii]

DeToledo, J. C., Lowe, M. R., & Puig, A. (2000). Nonepileptic seizures in pregnancy. *Neurology*, 55(1), 120-121.

Devinsky, O. (1997). Neurological aspects of the conscious and unconscious mind. *Annals of the New York Academy of Sciences*, 835(1), 321-329.



Devinsky, O., & Fisher, R. (1996). Ethical use of placebos and provocative testing in diagnosing nonepileptic seizures. *Neurology*, 47(4), 866-870.

Devinsky, O., Gazzola, D., & LaFrance, W. C. (2011). Differentiating between nonepileptic and epileptic seizures. *Nature Reviews Neurology*, 7(4), 210-220.

Devinsky, O., & Gordon, E. (1998). Epileptic seizures progressing into nonepileptic conversion seizures. *Neurology*, 51(5), 1293-1296.

Devinsky, O., Mesad, S., & Alper, K. (2001). Nondominant hemisphere lesions and conversion nonepileptic seizures. *Journal of Neuropsychiatry and Clinical Neurosciences*, 13(3), 367-373. doi:<http://dx.doi.org/10.1176/appi.neuropsych.13.3.367>

Devinsky, O., Sanchez-Villasenor, F., Vazquez, B., Kothari, M., Alper, K., & Luciano, D. (1996). Clinical profile of patients with epileptic and nonepileptic seizures. *Neurology*, 46(6), 1530-1533.

Devireddy, V. K., & Sharma, A. (2014). A case of psychogenic non-epileptic seizures, unresponsive type, in pregnancy. doi:<http://dx.doi.org/10.4088/PCC.13101574>

Dhiman, V., Sinha, S., Rawat, V. S., Harish, T., Chaturvedi, S. K., & Satishchandra, P. (2013). Semiological characteristics of adults with psychogenic nonepileptic seizures (PNESs): An attempt towards a new classification. *Epilepsy and Behavior*, 27(3), 427-432. doi:<http://dx.doi.org/10.1016/j.yebeh.2013.03.005>

Dhiman, V., Sinha, S., Rawat, V. S., Vijaysagar, K. J., Thippeswamy, H., Srinath, S., . . . Satishchandra, P. (2013). Children with psychogenic non-epileptic seizures (PNES): A detailed semiologic analysis and modified new classification. *Brain & Development*, 36 (4), 287-293. doi:10.1016/j.braindev.2013.05.002; 10.1016/j.braindev.2013.05.002

Dickinson, P., Looper, K. J., & Groleau, D. (2011). Patients diagnosed with nonepileptic seizures: Their perspectives and experiences. *Epilepsy & Behavior*, 20(3), 454-461. doi:<http://dx.doi.org/10.1016/j.yebeh.2010.12.034>

- Dikel, T. N., Fennell, E. B., & Gilmore, R. L. (2003). Posttraumatic stress disorder, dissociation, and sexual abuse history in epileptic and nonepileptic seizure patients. *Epilepsy & Behavior*, 4(6), 644-650. doi:10.1016/j.yebeh.2003.08.006
- Dimaro, L. V., Dawson, D. L., Roberts, N. A., Brown, I., Moghaddam, N. G., & Reuber, M. (2014). Anxiety and avoidance in psychogenic nonepileptic seizures: The role of implicit and explicit anxiety. *Epilepsy & Behavior*, 33(0), 77-86. doi:http://dx.doi.org/10.1016/j.yebeh.2014.02.016
- Ding, J., An, D., Liao, W., Li, J., Wu, G., Xu, Q., . . . Chen, H. (2013). Altered functional and structural connectivity networks in psychogenic non-epileptic seizures. *Plos One*, 8(5), e63850. doi:10.1371/journal.pone.0063850
- Ding, J., An, D., Liao, W., Wu, G., Xu, Q., Zhou, D., & Chen, H. (2014). Abnormal functional connectivity density in psychogenic non-epileptic seizures. *Epilepsy Research*, 108(7), 1184-1194. doi:http://dx.doi.org/10.1016/j.eplepsyres.2014.05.006
- Direk, N., Kulaksizoglu, I. B., Alpay, K., & Gurses, C. (2012). Using personality disorders to distinguish between patients with psychogenic nonepileptic seizures and those with epileptic seizures. *Epilepsy and Behavior*, 23(2), 138-141. doi:http://dx.doi.org/10.1016/j.yebeh.2011.11.013
- DiTomasso, M. J., & Routh, D. K. (1993). Recall of abuse in childhood and three measures of dissociation. *Child Abuse & Neglect*, 17(4), 477-485.
- Dixit, R., Popescu, A., Bagic, A., Ghearing, G., & Hendrickson, R. (2013). Medical comorbidities in patients with psychogenic nonepileptic spells (PNES) referred for video-EEG monitoring. *Epilepsy & Behavior*, 28(2), 137-140. doi:10.1016/j.yebeh.2013.05.004
- Dodrill, C. B. (2008). Do patients with psychogenic nonepileptic seizures produce trustworthy findings on neuropsychological tests? *Epilepsia*, 49(4), 691-695. doi:10.1111/j.1528-1167.2007.01457.x

Domes, G., Czeschnek, D., Weidler, F., Berger, C., Fast, K., & Herpertz, S. C. (2008). Recognition of facial affect in borderline personality disorder. *Journal of Personality Disorders*, 22(2), 135-147.

Dorahy, M. J., Middleton, W., & Irwin, H. J. (2005). The effect of emotional context on cognitive inhibition and attentional processing in dissociative identity disorder. *Behaviour Research and Therapy*, 43(5), 555-568.

Draijer, N., & Langeland, W. (1999). Childhood trauma and perceived parental dysfunction in the etiology of dissociative symptoms in psychiatric inpatients. *The American Journal of Psychiatry*, 156(3), 379-385.

Drake, Jr, M.E., Huber, S. J., Pakalnis, A., & Phillips, B. B. (1993). Neuropsychological and event related potential correlates of nonepileptic seizures. *Journal of Neuropsychiatry and Clinical Neurosciences*, 5(1), 102-104.

Drake Jr, M. E., Pakalnis, A., & Phillips, B. B. (1992). Neuropsychological and psychiatric correlates of intractable pseudoseizures. *Seizure*, 1(1), 11-13.  
doi:[http://dx.doi.org/10.1016/1059-1311\(92\)90048-6](http://dx.doi.org/10.1016/1059-1311(92)90048-6)

Drane, D. L., & Meador, K. J. (2002). Cognitive and behavioral effects of antiepileptic drugs. *Epilepsy & Behavior*, 3(5), 49-53.

Drane, D. L., Williamson, D. J., Stroup, E. S., Holmes, M. D., Jung, M., Koerner, E., . . . Miller, J. W. (2006). Cognitive impairment is not equal in patients with epileptic and psychogenic nonepileptic seizures. *Epilepsia*, 47(11), 1879-1886. doi:10.1111/j.1528-1167.2006.00611.x

Driver-Dunckley, E., Stonnington, C. M., Locke, D. E. C., & Noe, K. (2011). Comparison of psychogenic movement disorders and psychogenic nonepileptic seizures: Is phenotype clinically important? *Psychosomatics*, 52(4), 337-345.  
doi:<http://dx.doi.org/10.1016/j.psych.2011.01.008>

Duncan, R. (2010). Psychogenic nonepileptic seizures: Diagnosis and initial management. *Expert Review of Neurotherapeutics*, 10(12), 1803-1809.

doi:<http://dx.doi.org/10.1586/ern.10.171>

Duncan, R., Graham, C. D., & Oto, M. (2014a). Neurologist assessment of reactions to the diagnosis of psychogenic nonepileptic seizures: Relationship to short- and long-term outcomes. *Epilepsy & Behavior*, 41(0), 79-82.

doi:<http://dx.doi.org/10.1016/j.yebeh.2014.09.037>

Duncan, R., Graham, C. D., & Oto, M. (2014b). Outcome at 5–10 years in psychogenic nonepileptic seizures: What patients report vs. what family doctors report. *Epilepsy & Behavior*, 37(0), 71-74. doi:<http://dx.doi.org/10.1016/j.yebeh.2014.06.011>

Duncan, R., & Oto, M. (2008). Predictors of antecedent factors in psychogenic nonepileptic attacks: Multivariate analysis. *Neurology*, 71(13), 1000-1005.

doi:10.1212/01.wnl.0000326593.50863.21

Duncan, R., Oto, M., Martin, E., & Pelosi, A. (2006). Late onset psychogenic nonepileptic attacks. *Neurology*, 66(11), 1644-1647.

doi:<http://dx.doi.org/10.1212/01.wnl.0000223320.94812.7a>

Duncan, R., Razvi, S., & Mulhern, S. (2011). Newly presenting psychogenic nonepileptic seizures: Incidence, population characteristics, and early outcome from a prospective audit of a first seizure clinic. *Epilepsy & Behavior*, 20(2), 308-311.

Durrant, J., Rickards, H., & Cavanna, A. E. (2011). Prognosis and outcome predictors in psychogenic nonepileptic seizures. *Epilepsy Research and Treatment*, 2011, 274736-274736, doi:<http://dx.doi.org/10.1155/2011/274736>

Dworetzky, B. A., Bubrick, E. J., & Szaflarski, J. P. (2010). Nonepileptic psychogenic status: Markedly prolonged psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 19(1), 65-68.

Dworetzky, B. A., Mortati, K. A., Rossetti, A. O., Vaccaro, B., Nelson, A., & Bromfield, E. B. (2006). Clinical characteristics of psychogenic nonepileptic seizure status in the long-term monitoring unit. *Epilepsy & Behavior*, 9(2), 335-338.

Dworetzky, B. A., Strahonja-Packard, A., Shanahan, C. W., Paz, J., Schauble, B., & Bromfield, E. B. (2005). Characteristics of male veterans with psychogenic nonepileptic seizures. *Epilepsia*, 46(9), 1418-1422.

Dyck, M., Habel, U., Slodczyk, J., Schlummer, J., Backes, V., Schneider, F., & Reske, M. (2009). Negative bias in fast emotion discrimination in borderline personality disorder. *Psychological Medicine*, 39(5), 855-864.

Eastwood, J. D., Smilek, D., & Merikle, P. M. (2003). Negative facial expression captures attention and disrupts performance. *Perception & Psychophysics*, 65(3), 352-358.

Ebner-Priemer, U. W., Badeck, S., Beckmann, C., Wagner, A., Feige, B., Weiss, I., . . . Bohus, M. (2005). Affective dysregulation and dissociative experience in female patients with borderline personality disorder: A startle response study. *Journal of Psychiatric Research*, 39(1), 85-92.

Eddy, C. M., & Cavanna, A. E. (2014). Video-electroencephalography investigation of ictal alterations of consciousness in epilepsy and nonepileptic attack disorder: Practical considerations. *Epilepsy & Behavior*, 30(0), 24-27.  
doi:<http://dx.doi.org/10.1016/j.yebeh.2013.09.003>

Ekman, P. (1992). An argument for basic emotions. *Cognition & Emotion*, 6(3-4), 169-200.

Ekman, P., & Friesen, W. V. (1971). Constants across cultures in the face and emotion. *Journal of Personality and Social Psychology*, 17(2), 124.

Ekman, P., & Friesen, W. V. (1976). *Pictures of facial affect*. Palo Alto, CA: Consulting Psychologists Press.

Elfenbein, H. A., & Ambady, N. (2002). On the universality and cultural specificity of emotion recognition: A meta-analysis. *Psychological Bulletin*, 128(2), 203.

Elliott, J. O., & Charyton, C. (2014). Biopsychosocial predictors of psychogenic non-epileptic seizures. *Epilepsy Research*, 108(9), 1543-1553.

doi:<http://dx.doi.org/10.1016/j.eplepsyres.2014.09.003>

Elzinga, B. M., Schmahl, C. G., Vermetten, E., van Dyck, R., & Bremner, J. D. (2003). Higher cortisol levels following exposure to traumatic reminders in abuse-related PTSD. *Neuropsychopharmacology*, 28(9), 1656-1665.

Engel Jr., C. C., Walker, E. A., & Katon, W. J. (1996). Factors related to dissociation among patients with gastrointestinal complaints. *Journal of Psychosomatic Research*, 40(6), 643-653. doi:[http://dx.doi.org/10.1016/0022-3999\(95\)00636-2](http://dx.doi.org/10.1016/0022-3999(95)00636-2)

Esteves, F., Dimberg, U., & Öhman, A. (1994). Automatically elicited fear: Conditioned skin conductance responses to masked facial expressions. *Cognition & Emotion*, 8(5), 393-413.

Esteves, F., & Öhman, A. (1993). Masking the face: Recognition of emotional facial expressions as a function of the parameters of backward masking. *Scandinavian Journal of Psychology*, 34(1), 1-18.

Ettinger, A. B., Devinsky, O., Weisbrot, D. M., Ramakrishna, R. K., & Goyal, A. (1999). A comprehensive profile of clinical, psychiatric, and psychosocial characteristics of patients with psychogenic nonepileptic seizures. *Epilepsia*, 40(9), 1292-1298.

doi:<http://dx.doi.org/10.1111/j.1528-1157.1999.tb00860.x>

Ettinger, A. B., Dhoon, A., Weisbrot, D. M., & Devinsky, O. (1999). Predictive factors for outcome of nonepileptic seizures after diagnosis. *Journal of Neuropsychiatry and Clinical Neurosciences*, 11(4), 458-463.

Ettinger, A. B., Weisbrot, D. M., Nolan, E., & Devinsky, O. (1999). Postictal symptoms help distinguish patients with epileptic seizures from those with non-epileptic seizures. *Seizure*, 8(3), 149-151. doi:<http://dx.doi.org/10.1053/seiz.1999.0270>

Faber, D. P. (1997). Jean-Martin Charcot and the epilepsy/hysteria relationship. *Journal of the History of the Neurosciences*, 6(3), 275-290.

Fairclough, G., Fox, J., Mercer, G., Reuber, M., & Brown, R. J. (2014). Understanding the perceived treatment needs of patients with psychogenic nonepileptic seizures. *Epilepsy and Behavior*, 31, 295-303. doi:<http://dx.doi.org/10.1016/j.yebeh.2013.10.025>

Fargo, J. D., Schefft, B. K., Szaflarski, J. P., Dulay, M. F., Testa, S. M., Privitera, M. D., & Yeh, H. S. (2004). Accuracy of self-reported neuropsychological functioning in individuals with epileptic or psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 5(2), 143-150. doi:10.1016/j.yebeh.2003.11.023

Farias, S. T., Thieman, C., & Alsaadi, T. M. (2003). Psychogenic nonepileptic seizures: Acute change in event frequency after presentation of the diagnosis. *Epilepsy & Behavior*, 4(4), 424-429. doi:[http://dx.doi.org/10.1016/S1525-5050\(03\)00143-4](http://dx.doi.org/10.1016/S1525-5050(03)00143-4)

Farnam, A., Goreishizadeh, M. A., Farhang, S., & Abdolaliyan, F. (2008). High prevalence of depression among Iranian patients with first onset pseudoseizures. *Indian Journal of Psychiatry*, 50(4), 266-268. doi:10.4103/0019-5545.44749 [doi]

Faul, F., Erdfelder, E., Buchner, A., & Lang, A. (2009). Statistical power analyses using G\* power 3.1: Tests for correlation and regression analyses. *Behavior Research Methods*, 41(4), 1149-1160.

Faul, F., Erdfelder, E., Lang, A., & Buchner, A. (2007). G\* power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, 39(2), 175-191.

Feinstein, A., Stergiopoulos, V., Fine, J., & Lang, A. E. (2001). Psychiatric outcome in patients with a psychogenic movement disorder: A prospective study. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, 14(3), 169-176.

Fejerman, N. (2005). Nonepileptic disorders imitating generalized idiopathic epilepsies. *Epilepsia*, 46 Suppl 9, 80-83. doi:10.1111/j.1528-1167.2005.00317.x

Feliu-Soler, A., Pascual, J. C., Soler, J., Pérez, V., Armario, A., Carrasco, J., . . . Borràs, X. (2013). Emotional responses to a negative emotion induction procedure in borderline personality disorder. *International Journal of Clinical and Health Psychology*, 13(1), 9-17.

Field, A. (2013). *Discovering statistics using IBM SPSS statistics* (4th ed.). London: Sage.

Fizman, A., Alves-Leon, S. V., Nunes, R. G., D'Andrea, I., & Figueira, I. (2004). Traumatic events and posttraumatic stress disorder in patients with psychogenic nonepileptic seizures: A critical review. *Epilepsy & Behavior*, 5(6), 818-825. doi:10.1016/j.yebeh.2004.09.002

Fizman, A., & Kanner, A. M. (2010). Comorbidities in psychogenic nonepileptic seizures: Depressive, anxiety, and personality disorders. In S. C. Schachter, & W. C. LaFrance Jr (Eds.), *Gates and Rowan's nonepileptic seizures* (3rd ed., pp. 225-236). Cambridge: Cambridge University Press.

Fleisher, W., Staley, D., Krawetz, P., Pillay, N., Arnett, J. L., & Maher, J. (2002). Comparative study of trauma-related phenomena in subjects with pseudoseizures and subjects with epilepsy. *American Journal of Psychiatry*, 159(4), 660-663. doi:10.1176/appi.ajp.159.4.660

Flugel, D., Bauer, J., Kaseborn, U., Burr, W., & Elger, C. E. (1996). Closed eyes during a seizure indicate psychogenic etiology: A study with suggestive seizure provocation. *Journal of Epilepsy*, 9(3), 165-169. doi:10.1016/0896-6974(96)00008-4



Foa, E. B., Cashman, L., Jaycox, L., & Perry, K. (1997). The validation of a self-report measure of posttraumatic stress disorder: The posttraumatic diagnostic scale. *Psychological Assessment*, 9(4), 445.

Foa, E. B., & Hearst-Ikeda, D. (1996). Emotional dissociation in response to trauma: An information processing approach. In L. K. Michelson, & W. J. Ray (Eds.), *Handbook of dissociation: Theoretical, empirical, and clinical perspectives* (pp. 207-224). New York: Plenum Press.

Foote, B., Smolin, Y., Kaplan, M., Legatt, M. E., & Lipschitz, D. (2006). Prevalence of dissociative disorders in psychiatric outpatients. *The American Journal of Psychiatry*, 163(4), 623-629. doi:10.1176/ajp.163.4.623 [pii]

Frances, P. L., Baker, G. A., & Appleton, P. L. (1999). Stress and avoidance in pseudoseizures: Testing the assumptions. *Epilepsy Research*, 34(2-3), 241-249. doi:10.1016/S0920-1211(98)00116-8

Francis, P. A., & Baker, G. A. (1999). Non-epileptic attack disorder (NEAD): A comprehensive review. *Seizure*, 8(1), 53-61. doi:10.1053/seiz.1998.0246

French, J. (1995). Pseudoseizures in the era of video-electroencephalogram monitoring. *Current Opinion in Neurology*, 8(2), 117-120.

Frith, C., & Rees, G. A brief history of the scientific approach to the study of consciousness. In M. Velmans, & S. Schneider (Eds.), *The Blackwell Companion to Consciousness* (pp. 9-22). Oxford: Blackwell Publishing Ltd.

Fritzsche, K., Baumann, K., Gotz-Trabert, K., & Schulze-Bonhage, A. (2013). Dissociative seizures: A challenge for neurologists and psychotherapists. *Deutsches Arzteblatt International*, 110(15), 263-268. doi:http://dx.doi.org/10.3238/arztebl.2013.0263

- Frolov, M. V., Korinevskaya, I. V., & Vorob'eva, O. V. (2003). Phenomenological assessment of psychogenic paroxysms using parameters of verbal activity. *Fiziologiya Cheloveka*, 29(1), 32-37.
- Gale, S. D., & Hill, S. W. (2012). Concurrent administration of the MMPI-2 and PAI in a sample of patients with epileptic or non-epileptic seizures: Implications for an inpatient epilepsy monitoring unit. *Epilepsy and Behavior*, 25(2), 181-184.  
doi:<http://dx.doi.org/10.1016/j.yebeh.2012.07.012>
- Galimberti, C. A., Ratti, M. T., Murelli, R., Marchioni, E., Manni, R., & Tartara, A. (2003). Patients with psychogenic nonepileptic seizures, alone or epilepsy-associated, share a psychological profile distinct from that of epilepsy patients. *Journal of Neurology*, 250(3), 338-346. doi:10.1007/s00415-003-1009-0
- Gambini, O., Demartini, B., Chiesa, V., Turner, K., Barbieri, V., & Canevini, M. P. (2014). Long-term outcome of psychogenic nonepileptic seizures: The role of induction by suggestion. *Epilepsy & Behavior*, 41(0), 140-143.  
doi:<http://dx.doi.org/10.1016/j.yebeh.2014.09.076>
- Garnefski, N., & Kraaij, V. (2007). The cognitive emotion regulation questionnaire. *European Journal of Psychological Assessment*, 23(3), 141-149.
- Gates, J. R. (2000). Nonepileptic seizures: Time for progress. *Epilepsy & Behavior*, 1(1), 2-6. doi:<http://dx.doi.org/10.1006/ebbeh.2000.0029>
- Gates, J. R. (2001). Provocative testing should not be used for nonepileptic seizures. *Archives of Neurology*, 58(12), 2065-2066.
- Gates, J. R. (2002). Nonepileptic seizures: Classification, coexistence with epilepsy, diagnosis, therapeutic approaches, and consensus. *Epilepsy and Behavior*, 3(1), 28-33.  
doi:<http://dx.doi.org/10.1006/ebbeh.2001.0310>
- Gates, J. R., Ramani, V., Whalen, S., & Loewenson, R. (1985). Ictal characteristics of pseudoseizures. *Archives of Neurology*, 42(12), 1183-1187.

Gazzola, D. M., Carlson, C., Rugino, A., Hirsch, S., Starner, K., & Devinsky, O. (2012). Psychogenic nonepileptic seizures and chronic pain: A retrospective case-controlled study. *Epilepsy and Behavior*, 25(4), 662-665.

doi:<http://dx.doi.org/10.1016/j.yebeh.2012.10.008>

Gene-Cos, N., Pottinger, R., Barrett, G., Trimble, M. R., & Ring, H. A. (2005). A comparative study of mismatch negativity (MMN) in epilepsy and non-epileptic seizures. *Epileptic Disorders*, 7(4), 363-372.

Gershuny, B. S., & Thayer, J. F. (1999). Relations among psychological trauma, dissociative phenomena, and trauma-related distress: A review and integration. *Clinical Psychology Review*, 19(5), 631-657.

Ghaffar, O., Staines, W. R., & Feinstein, A. (2006). Unexplained neurologic symptoms: An fMRI study of sensory conversion disorder. *Neurology*, 67(11), 2036-2038.

doi:67/11/2036 [pii]

Glass, G. V., Peckham, P. D., & Sanders, J. R. (1972). Consequences of failure to meet assumptions underlying the fixed effects analyses of variance and covariance. *Review of Educational Research*, , 237-288.

Glosser, G., Roberts, D., & Glosser, D. S. (1999). Nonepileptic seizures after resective epilepsy surgery. *Epilepsia*, 40(12), 1750-1754.

Golden, C. J. (1975). A group version of the Stroop color and word test. *Journal of Personality Assessment*, 39(4), 386-388.

Golden, C. J., & Freshwater, S. M. (2002). *The Stroop color and word test: A manual for clinical and experimental uses*. Chicago, IL: Stoelting.

Goldstein, L. H., Chalder, T., Chigwedere, C., Khondoker, M. R., Moriarty, J., Toone, B. K., & Mellers, J. D. C. (2010). Cognitive-behavioral therapy for psychogenic nonepileptic seizures A pilot RCT. *Neurology*, 74(24), 1986-1994.

Goldstein, L. H., Chalder, T., Chigwedere, C., Moriarty, J., Toone, B. K., & Mellers, J. D. (2009). A randomised controlled trial of CBT and standard medical care in patients with dissociative (non-epileptic) seizures. *Epilepsia*, 50, 221-222.

Goldstein, L. H., Deale, A. C., Mitchell-O'Malley, S. J., Toone, B. K., & Mellers, J. D. C. (2004). An evaluation of cognitive behavioral therapy as a treatment for dissociative seizures: A pilot study. *Cognitive and Behavioral Neurology*, 17(1), 41-49.  
doi:<http://dx.doi.org/10.1097/00146965-200403000-00005>

Goldstein, L. H., Drew, C., Mellers, J., Mitchell-O'Malley, S., & Oakley, D. A. (2000). Dissociation, hypnotizability, coping styles and health locus of control: Characteristics of pseudoseizure patients. *Seizure*, 9(5), 314-322. doi:10.1053/seiz.2000.0421

Goldstein, L. H., & Mellers, J. D. C. (2006). Ictal symptoms of anxiety, avoidance behaviour, and dissociation in patients with dissociative seizures. *Journal of Neurology Neurosurgery and Psychiatry*, 77(5), 616-621. doi:10.1136/jnnp.2005.066878

Goldstein, L. H., & Mellers, J. D. C. (2012). Recent developments in our understanding of the semiology and treatment of psychogenic nonepileptic seizures. *Current Neurology and Neuroscience Reports*, 12(4), 436-444. doi:<http://dx.doi.org/10.1007/s11910-012-0278-3>

Goodwin, J. M., & Sachs, R. G. (1996). Child abuse in the etiology of dissociative disorders. In L. K. Michelson, & W. J. Ray (Eds.), *Handbook of dissociation: Theoretical, empirical, and clinical perspectives* (pp. 91-105). New York: Plenum Press.

Goyal, G., Kalita, J., & Misra, U. K. (2014). Utility of different seizure induction protocols in psychogenic nonepileptic seizures. *Epilepsy Research*, 108(6), 1120-1127. doi:<http://dx.doi.org/10.1016/j.eplepsyres.2014.02.015>

Green, A., Payne, S., & Barnitt, R. (2004). Illness representations among people with non-epileptic seizures attending a neuropsychiatry clinic: A qualitative study based on the self-regulation model. *Seizure*, 13(5), 331-339. doi:10.1016/j.seizure.2003.09.001

- Griffith, J. L., Polles, A., & Griffith, M. E. (1998). Pseudoseizures, families, and unspeakable dilemmas. *Psychosomatics*, 39(2), 144-153.
- Griffith, N. M., & Szaflarski, J. P. (2010). Epidemiology and classification of psychogenic nonepileptic seizures. In S. C. Schachter, & W. C. LaFrance Jr (Eds.), *Gates and Rowan's nonepileptic seizures* (3rd ed., pp. 3-16). Cambridge: Cambridge University Press.
- Griffith, N. M., Szaflarski, J. P., Schefft, B. K., Isaradisakul, D., Meckler, J. M., McNally, K. A., & Privitera, M. D. (2007). Relationship between semiology of psychogenic nonepileptic seizures and Minnesota Multiphasic Personality Inventory profile. *Epilepsy and Behavior*, 11(1), 105-111. doi:<http://dx.doi.org/10.1016/j.yebeh.2007.04.021>
- Grimaldi, I., Dubuc, M., Kahane, P., Bougerol, T., & Vercueil, L. (2010). Anxiety and depression in psychogenic movement disorder and non-epileptic seizures: A prospective comparative study. *Revue Neurologique*, 166(5), 515-522. doi:10.1016/j.neurol.2009.10.016
- Gröppel, G., Kapitany, T., & Baumgartner, C. (2000). Cluster analysis of clinical seizure semiology of psychogenic nonepileptic seizures. *Epilepsia*, 41(5), 610-614.
- Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology*, 85(2), 348.
- Gudmundsson, O., Prendergast, M., Foreman, D., & Cowley, S. (2001). Outcome of pseudoseizures in children and adolescents: A 6-year symptom survival analysis. *Developmental Medicine and Child Neurology*, 43(8), 547-551. doi:10.1017/S0012162201000986
- Gul, A., & Ahmad, H. (2014). Cognitive deficits and emotion regulation strategies in patients with psychogenic nonepileptic seizures: A task-switching study. *Epilepsy & Behavior*, 32(0), 108-113. doi:<http://dx.doi.org/10.1016/j.yebeh.2014.01.015>

- Guz, H., Doganay, Z., Ozkan, A., Colak, E., Tomac, A., & Sarisoy, G. (2003). Conversion disorder and its subtypes: A need for a reclassification. *Nordic Journal of Psychiatry*, 57(5), 377-381. doi:10.1080/08039480310002723
- Hall, J. A., & Matsumoto, D. (2004). Gender differences in judgments of multiple emotions from facial expressions. *Emotion*, 4(2), 201.
- Halligan, P. W., Bass, C., & Wade, D. T. (2000). New approaches to conversion hysteria. *BMJ (Clinical Research Ed.)*, 320(7248), 1488-1489.
- Hallion, L. S., & Ruscio, A. M. (2011). A meta-analysis of the effect of cognitive bias modification on anxiety and depression. *Psychological Bulletin*, 137(6), 940.
- Hall-Patch, L., Brown, R., House, A., Howlett, S., Kemp, S., Lawton, G., . . . Reuber, M. (2010). Acceptability and effectiveness of a strategy for the communication of the diagnosis of psychogenic nonepileptic seizures. *Epilepsia*, 51(1), 70-78. doi:http://dx.doi.org/10.1111/j.1528-1167.2009.02099.x
- Harden, C. L. (1997). Pseudoseizures and dissociative disorders: A common mechanism involving traumatic experiences. *Seizure*, 6(2), 151-155. doi:10.1016/S1059-1311(97)80070-2
- Harden, C. L., Jovine, L., Burgut, F. T., Carey, B. T., Nikolov, B. G., & Ferrando, S. J. (2009). A comparison of personality disorder characteristics of patients with nonepileptic psychogenic pseudoseizures with those of patients with epilepsy. *Epilepsy & Behavior*, 14(3), 481-483. doi:http://dx.doi.org/10.1016/j.yebeh.2008.12.012
- Harmer, C.J., Bhagwagar, Z., Perrett, D., Vollm, B., Cowen, P., & Goodwin, G. (2003). Acute SSRI administration affects the processing of social cues in healthy volunteers. *Neuropsychopharmacology*, 28(1), 148-152.
- Harmer, C. J., Mackay, C. E., Reid, C. B., Cowen, P. J., & Goodwin, G. M. (2006). Antidepressant drug treatment modifies the neural processing of nonconscious threat cues. *Biological Psychiatry*, 59(9), 816-820.

Harrison, A., Sullivan, S., Tchanturia, K., & Treasure, J. (2010). Emotional functioning in eating disorders: Attentional bias, emotion recognition and emotion regulation. *Psychological Medicine*, 40(11), 1887-1897.

Hathaway, S. R., & McKinley, J. C. (1943). *The Minnesota multiphasic personality inventory (MMPI)*. Minneapolis: University of Minnesota Press.

Hathaway, S. R., McKinley, J. C., & MMPI Restandardization Committee. (1989). *MMPI-2: Minnesota multiphasic personality inventory-2: Manual for administration and scoring*. Minneapolis: University of Minnesota Press.

Hayes, A. F. (2012). *PROCESS: A versatile computational tool for observed variable mediation, moderation, and conditional process modeling*. Retrieved from <http://www.afhayes.com/public/process2012.pdf>.

Hendrickson, R., Popescu, A., Dixit, R., Ghearing, G., & Bagic, A. (2014). Panic attack symptoms differentiate patients with epilepsy from those with psychogenic nonepileptic spells (PNES). *Epilepsy & Behavior*, 37(0), 210-214.  
doi:<http://dx.doi.org/10.1016/j.yebeh.2014.06.026>

Hermans, E. J., Nijenhuis, E. R., Van Honk, J., Huntjens, R. J., & van der Hart, O. (2006). Identity state-dependent attentional bias for facial threat in dissociative identity disorder. *Psychiatry Research*, 141(2), 233-236.

Herpertz, S. C., Kunert, H. J., Schwenger, U. B., & Sass, H. (1999). Affective responsiveness in borderline personality disorder: A psychophysiological approach. *The American Journal of Psychiatry*, 156(10), 1550-1556.

Hilgard, E. R. (1994). Neodissociation theory. In S. J. Lynn, & J. W. Rhue (Eds.), *Dissociation: Clinical and theoretical perspectives* (pp. 32-51). New York: The Guildford Press.

Hill, S. W., & Gale, S. D. (2011a). Neuropsychological characteristics of nonepileptic seizure semiological subgroups. *Epilepsy & Behavior*, 22(2), 255-260.

Hill, S. W., & Gale, S. D. (2011b). Predicting psychogenic nonepileptic seizures with the personality assessment inventory and seizure variables. *Epilepsy & Behavior*, 22(3), 505-510. doi:<http://dx.doi.org/10.1016/j.yebeh.2011.08.001>

Hines, E. A., & Brown, G. E. (1936). The cold pressor test for measuring the reactivity of the blood pressure: Data concerning 571 normal and hypertensive subjects. *American Heart Journal*, 11(1), 1-9.

Hingray, C., Maillard, L., Hubsch, C., Vignal, J., Bourgognon, F., Laprevote, V., . . . Schwan, R. (2011). Psychogenic nonepileptic seizures: Characterization of two distinct patient profiles on the basis of trauma history. *Epilepsy & Behavior*, 22(3), 532-536. doi:10.1016/j.yebeh.2011.08.015

Hixson, J. D., Balcer, L. J., Glosser, G., & French, J. A. (2006). Fear sensitivity and the psychological profile of patients with psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 9(4), 587-592. doi:10.1016/j.yebeh.2006.08.002

Hixson, J. D., & Kirsch, H. E. (2009). The effects of epilepsy and its treatments on affect and emotion. *Neurocase*, 15(3), 206-216.

Hochberg, Y. (1988). A sharper Bonferroni procedure for multiple tests of significance. *Biometrika*, 75(4), 800-802.

Hoepner, R., Labudda, K., May, T. W., Schondienst, M., Bien, C. G., & Brandt, C. (2014). Distinguishing between patients with pure psychogenic nonepileptic seizures and those with comorbid epilepsy by means of clinical data. *Epilepsy and Behavior*, 35, 54-58. doi:<http://dx.doi.org/10.1016/j.yebeh.2014.04.002>

Hoepner, R., Labudda, K., Schoendienst, M., May, T. W., Bien, C. G., & Brandt, C. (2013). Informing patients about the impact of provocation methods increases the rate of psychogenic nonepileptic seizures during EEG recording. *Epilepsy and Behavior*, 28(3), 457-459. doi:<http://dx.doi.org/10.1016/j.yebeh.2013.06.009>



Hoerth, M. T., Wellik, K. E., Demaerschalk, B. M., Dratzkowski, J. F., Noe, K. H., Sirven, J. I., & Wingerchuk, D. M. (2008). Clinical predictors of psychogenic nonepileptic seizures: A critically appraised topic. *The Neurologist*, 14(4), 266-270.

doi:10.1097/NRL.0b013e31817acee4; 10.1097/NRL.0b013e31817acee4

Holman, N., Kirkby, A., Duncan, S., & Brown, R. J. (2008). Adult attachment style and childhood interpersonal trauma in non-epileptic attack disorder. *Epilepsy Research*, 79(1), 84-89. doi:10.1016/j.epilepsyres.2007.12.015

Holmes, E. A., Brown, R. J., Mansell, W., Fearon, R., Hunter, E., Frasquilho, F., & Oakley, D. A. (2005). Are there two qualitatively distinct forms of dissociation? A review and some clinical implications. *Clinical Psychology Review*, 25(1), 1-23.

Holmes, G. L., Sackellares, J. C., McKiernan, J., Ragland, M., & Dreifuss, F. E. (1980). Evaluation of childhood pseudoseizures using EEG telemetry and video tape monitoring. *The Journal of Pediatrics*, 97(4), 554-558.

Holmes, L. B., Harvey, E. A., Coull, B. A., Huntington, K. B., Khoshbin, S., Hayes, A. M., & Ryan, L. M. (2001). The teratogenicity of anticonvulsant drugs. *New England Journal of Medicine*, 344(15), 1132-1138.

Holmes, M. D., Dodrill, C. B., Bachtler, S., Wilensky, A. J., Ojemann, L. M., & Miller, J. W. (2001). Evidence that emotional maladjustment is worse in men than in women with psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 2(6), 568-573.  
doi:10.1006/ebch.2001.0268

Hopp, J. L., Anderson, K. E., Krumholz, A., Gruber-Baldini, A. L., & Shulman, L. M. (2012). Psychogenic seizures and psychogenic movement disorders: Are they the same patients? *Epilepsy & Behavior*, 25(4), 666-669.  
doi:http://dx.doi.org/10.1016/j.yebeh.2012.10.007

Hoppe, C., & Elger, C. E. (2011). Depression in epilepsy: A critical review from a clinical perspective. *Nature Reviews Neurology*, 7(8), 462-472. doi:10.1038/nrneuro.2011.104

Hosmer, D. W., & Lemeshow, S. (2000). *Applied logistic regression* (2nd ed.). New Jersey: Wiley Online Library.

Hovorka, J., Nezadal, T., Herman, E., Nemcova, I., & Bajacek, M. (2007). Psychogenic non-epileptic seizures, prospective clinical experience: Diagnosis, clinical features, risk factors, psychiatric comorbidity, treatment outcome. *Epileptic Disorders*, 9, S52-S58. doi:10.1684/epd.2008.0156

Howell, D. (1997). *Statistical methods for psychology* (4th ed.). Belmont, CA: Wadsworth.

Howell, S. J., Owen, L., & Chadwick, D. W. (1989). Pseudostatus epilepticus. *The Quarterly Journal of Medicine*, 71(266), 507-519.

Howlett, S., & Reuber, M. (2009). An augmented model of brief psychodynamic interpersonal therapy for patients with nonepileptic seizures. *Psychotherapy*, 46(1), 125-138. doi:10.1037/a0015138

Hubsch, C., Baumann, C., & Maillard, L. (2010). Psychogenic non-epileptic seizures: Clinical classification based on the video-EEG analysis of 145 seizures. *Journal of Neurology*, 257, S176-S176.

Iriarte, J., Parra, J., Urrestarazu, E., & Kuyk, J. (2003). Controversies in the diagnosis and management of psychogenic pseudoseizures. *Epilepsy & Behavior*, 4(3), 354-359.

Irwin, H. J. (1996). Traumatic childhood events, perceived availability of emotional support, and the development of dissociative tendencies. *Child Abuse & Neglect*, 20(8), 701-707.

Irwin, H. J. (1999). Pathological and nonpathological dissociation: The relevance of childhood trauma. *The Journal of Psychology*, 133(2), 157-164.

Isaac, M., & Chand, P. K. (2006). Dissociative and conversion disorders: Defining boundaries. *Current Opinion in Psychiatry*, 19(1), 61-66. doi:10.1097/01.yco.0000194811.83720.69 [doi]

- Ito, M., Adachi, N., Okazaki, M., Kato, M., & Onuma, T. (2009). Evaluation of dissociative experiences and the clinical utility of the Dissociative Experience Scale in patients with coexisting epilepsy and psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 16(3), 491-494. doi:10.1016/j.yebeh.2009.08.017 [doi]
- Jain, U., Jain, J., Tiruveedhula, V., & Sharma, A. (2013). Psychogenic non-epileptic seizures in a second-trimester pregnant woman with a previous child loss. doi:http://dx.doi.org/10.4088/PCC.13101562
- Jawad, S. S. M., Jamil, N., Clarke, E. J., Lewis, A., Whitecross, S., & Richens, A. (1995). Psychiatric morbidity and psychodynamics of patients with convulsive pseudoseizures. *Seizure*, 4(3), 201-206. doi:http://dx.doi.org/10.1016/S1059-1311(05)80061-5
- Jedrzejczak, J., Owczarek, K., & Majkowski, J. (1999). Psychogenic pseudoepileptic seizures: Clinical and electroencephalogram (EEG) video-tape recordings. *European Journal of Neurology*, 6(4), 473-479.
- Jensen, A. R. (1965). Scoring the Stroop test. *Acta Psychologica*, 24, 398-408.
- Jimenez, X. F., Sharma, J. S., & Dar, S. A. (2014). Conversion disorder as psychogenic nonepileptic seizures in suspected cancer: A case report. *General Hospital Psychiatry*, 36(6), 761.e1-761.e2. doi:http://dx.doi.org/10.1016/j.genhosppsych.2014.06.012
- Jirsch, J., Ahmed, S. N., & Gross, D. W. (2011). Recognition of psychogenic nonepileptic seizures diminishes acute care utilization. *Epilepsy & Behavior*, 22(2), 304-307.
- Jones, S. G., O'Brien, T. J., Adams, S. J., Mocellin, R., Kilpatrick, C. J., Yerra, R., . . . Velakoulis, D. (2010). Clinical characteristics and outcome in patients with psychogenic nonepileptic seizures. *Psychosomatic Medicine*, 72(5), 487-497. doi:10.1097/PSY.0b013e3181d96550
- Jongsma, M. J., Mommers, J. M., Renier, W. O., & Meinardi, H. (1999). Follow-up of psychogenic, non-epileptic seizures: A pilot study - experience in a Dutch special centre for epilepsy. *Seizure*, 8(3), 146-148. doi:10.1053/seiz.1998.0247

Kalogjera-Sackellares, D., & Sackellares, J. C. (1997). Analysis of MMPI patterns in patients with psychogenic pseudoseizures. *Seizure*, 6(6), 419-427.

doi:[http://dx.doi.org/10.1016/S1059-1311\(97\)80015-5](http://dx.doi.org/10.1016/S1059-1311(97)80015-5)

Kalogjera-Sackellares, D., & Sackellares, J. C. (1999). Intellectual and neuropsychological features of patients with psychogenic pseudoseizures. *Psychiatry Research*, 86(1), 73-84.

doi:10.1016/S0165-1781(99)00016-5

Kanner, A. M., Parra, J., Frey, M., Stebbins, G., Pierre-Louis, S., & Iriarte, J. (1999).

Psychiatric and neurologic predictors of psychogenic pseudoseizure outcome.

*Neurology*, 53(5), 933-938.

Kanner, A. M., Schachter, S. C., Barry, J. J., Hersdorffer, D. C., Mula, M., Trimble, M., . . .

. Gilliam, F. (2012). Depression and epilepsy: Epidemiologic and neurobiologic perspectives that may explain their high comorbid occurrence. *Epilepsy & Behavior*,

24(2), 156-168. doi:10.1016/j.yebeh.2012.01.007

Kaplan, M. J., Dwivedi, A. K., Privitera, M. D., Isaacs, K., Hughes, C., & Bowman, M.

(2013). Comparisons of childhood trauma, alexithymia, and defensive styles in patients with psychogenic non-epileptic seizures vs. epilepsy: Implications for the etiology of

conversion disorder. *Journal of Psychosomatic Research*, 75(2), 142-146.

doi:10.1016/j.jpsychores.2013.06.005

Karakis, I., Montouris, G. D., Piperidou, C., Luciano, M. S., Meador, K. J., & Cole, A. J.

(2014). Patient and caregiver quality of life in psychogenic non-epileptic seizures compared to epileptic seizures. *Seizure*, 23(1), 47-54.

doi:<http://dx.doi.org/10.1016/j.seizure.2013.09.011>

Karam, C. (2010). Psychogenic nonepileptic "seizures" or "attacks"? It's not just semantics: Seizures. *Neurology*, 75(23), 2135-2135.

doi:10.1212/WNL.0b013e3181fe7922

Karterud, H. N., Knizek, B. L., & Nakken, K. O. (2010). Changing the diagnosis from epilepsy to PNES: Patients' experiences and understanding of their new diagnosis. *Seizure-European Journal of Epilepsy*, 19(1), 40-46. doi:10.1016/j.seizure.2009.11.001

Kelley, S. D., & Benbadis, S. (2007). Eye movement desensitization and reprocessing in the psychological treatment of trauma-based psychogenic non-epileptic seizures. *Clinical Psychology & Psychotherapy*, 14(2), 135-144. doi:10.1002/cpp.525

Kelley, S. D., Benbadis, S. R., & Adams, D. (2005). A community-based study of eye movement desensitization and reprocessing in the psychological treatment of trauma-based psychogenic seizures. *Epilepsia*, 46, 347-347.

Kellinghaus, C., Loddenkemper, T., Dinner, D. S., Lachhwani, D., & Luders, H. O. (2004). Non-epileptic seizures of the elderly. *Journal of Neurology*, 251(6), 704-709. doi:10.1007/s00415-004-0406-3

Kennedy, F., Clarke, S., Stopa, L., Bell, L., Rouse, H., Ainsworth, C., . . . Waller, G. (2004). Towards a cognitive model and measure of dissociation. *Journal of Behavior Therapy and Experimental Psychiatry*, 35(1), 25-48.

Khan, A. Y., Baade, L., Ablah, E., McNerney, V., Golewale, M. H., & Liow, K. (2009). Can hypnosis differentiate epileptic from nonepileptic events in the video/EEG monitoring unit? Data from a pilot study. *Epilepsy & Behavior*, 15(3), 314-317. doi:http://dx.doi.org/10.1016/j.yebeh.2009.04.004

Kihlstrom, J. F. (2005). Dissociative disorders. *Annual Review of Clinical Psychology*, 1, 227-253. doi:10.1146/annurev.clinpsy.1.102803.143925 [doi]

Kim, S. H., Kim, H., Lim, B. C., Chae, J., Kim, K. J., Hwang, Y. S., & Hwang, H. (2012). Paroxysmal nonepileptic events in pediatric patients confirmed by long-term video-EEG monitoring — single tertiary center review of 143 patients. *Epilepsy & Behavior*, 24(3), 336-340. doi:http://dx.doi.org/10.1016/j.yebeh.2012.03.022

Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The 'Trier Social Stress Test' - a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, 28(1-2), 76-81. doi:119004 [pii]

Koby, D. G., Zirakzadeh, A., Staab, J. P., Seime, R., Cha, S. S., Nelson, C. L., . . . Worrell, G. A. (2010). Questioning the role of abuse in behavioral spells and epilepsy. *Epilepsy & Behavior*, 19(4), 584-590. doi:10.1016/j.yebeh.2010.09.014

Kohler, C. G., Hoffman, L. J., Eastman, L. B., Healey, K., & Moberg, P. J. (2011). Facial emotion perception in depression and bipolar disorder: A quantitative review. *Psychiatry Research*, 188(3), 303-309.

Koster, E. H., Fox, E., & MacLeod, C. (2009). Introduction to the special section on cognitive bias modification in emotional disorders. *Journal of Abnormal Psychology*, 118(1), 1.

Kotagal, P., Costa, M., Wyllie, E., & Wolgamuth, B. (2002). Paroxysmal nonepileptic events in children and adolescents. *Pediatrics*, 110(4), e46.

Kotamäki, M. (1995). Smoking induced differences in autonomic responses in military pilot candidates. *Clinical Autonomic Research*, 5(1), 31-36.

Kozłowska, K. (2005). Healing the disembodied mind: Contemporary models of conversion disorder. *Harvard Review of Psychiatry*, 13(1), 1-13.

Krahn, L. E., Reese, M. M., Rummans, T. A., Peterson, G. C., Suman, V. J., Sharbrough, F. W., & Cascino, G. D. (1997). Health care utilization of patients with psychogenic nonepileptic seizures. *Psychosomatics*, 38(6), 535-542.

Kramer, U., Carmant, L., Riviello, J. J., Stauffer, A., Helmers, S. L., Mikati, M. A., & Holmes, G. L. (1995). Psychogenic seizures: Video telemetry observations in 27 patients. *Pediatric Neurology*, 12(1), 39-41.

- Krawetz, P., Fleisher, W., Pillay, N., Staley, D., Arnett, J., & Maher, J. (2001). Family functioning in subjects with pseudoseizures and epilepsy. *Journal of Nervous and Mental Disease*, 189(1), 38-43. doi:10.1097/00005053-200101000-00007
- Kret, M. E., & De Gelder, B. (2012). A review on sex differences in processing emotional signals. *Neuropsychologia*, 50(7), 1211-1221. doi:10.1016/j.neuropsychologia.2011.12.022 [doi]
- Krishnamoorthy, E. S., Brown, R. J., & Trimble, M. R. (2001). Personality and psychopathology in nonepileptic attack disorder and epilepsy: A prospective study. *Epilepsy & Behavior*, 2(5), 418-422. doi:10.1006/ebeh.2001.0233
- Krishnan, B., Faith, A., Vlachos, I., Roth, A., Williams, K., Noe, K., . . . Iasemidis, L. (2011). Resetting of brain dynamics: Epileptic versus psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 22, S74-S81. doi:10.1016/j.yebeh.2011.08.036
- Kristensen, O., & Alving, J. (1992). Pseudoseizures - risk-factors and prognosis - a case-control study. *Acta Neurologica Scandinavica*, 85(3), 177-180.
- Krystal, J. H., Bennett, A., Bremner, J. D., Southwick, S. M., & Charney, D. S. (1996). Recent developments in the neurobiology of dissociation. In L. K. Michelson, & W. J. Ray (Eds.), *Handbook of dissociation: Theoretical, empirical, and clinical perspectives* (pp. 163-190). New York: Plenum Press.
- Kutluay, E., Selwa, L., Minecan, D., Edwards, J., & Beydoun, A. (2010). Nonepileptic paroxysmal events in a pediatric population. *Epilepsy & Behavior*, 17(2), 272-275. doi:10.1016/j.yebeh.2009.12.020; 10.1016/j.yebeh.2009.12.020
- Kuyk, J., Jacobs, L. D., Aldenkamp, A. P., Meinardi, H., Spinhoven, P., & Vandyck, R. (1995). Pseudo-epileptic seizures - hypnosis as a diagnostic-tool. *Seizure*, 4(2), 123-128. doi:10.1016/S1059-1311(95)80091-3

Kuyk, J., Leijten, F., Meinardi, H., Spinhoven, P., & VanDyck, R. (1997). The diagnosis of psychogenic non-epileptic seizures: A review. *Seizure*, 6(4), 243-253. doi:10.1016/S1059-1311(97)80072-6

Kuyk, J., Siffels, M. C., Bakvis, P., & Swinkels, W. A. (2008). Psychological treatment of patients with psychogenic non-epileptic seizures: An outcome study. *Seizure*, 17(7), 595-603. doi:10.1016/j.seizure.2008.02.006; 10.1016/j.seizure.2008.02.006

Kuyk, J., Spinhoven, P., Boas, W. V., & Van Dyck, R. (1999). Dissociation in temporal lobe epilepsy and pseudo-epileptic seizure patients. *Journal of Nervous and Mental Disease*, 187(12), 713-720. doi:10.1097/00005053-199912000-00002

Kuyk, J., Spinhoven, P., & van Dyck, R. (1999). Hypnotic recall: A positive criterion in the differential diagnosis between epileptic and pseudoepileptic seizures. *Epilepsia*, 40(4), 485-491. doi:10.1111/j.1528-1157.1999.tb00745.x

Kuyk, J., Swinkels, W. A. M., & Spinhoven, P. (2003). Psychopathologies in patients with nonepileptic seizures with and without comorbid epilepsy: How different are they? *Epilepsy and Behavior*, 4(1), 13-18. doi:http://dx.doi.org/10.1016/S1525-5050%2802%2900683-2

Kuyk, J., van Dyck, R., & Spinhoven, P. (1996). The case for a dissociative interpretation of pseudoepileptic seizures. *Journal of Nervous and Mental Disease*, 184(8), 468-474. doi:10.1097/00005053-199608000-00003

Labate, A., Cerasa, A., Mula, M., Mumoli, L., Gioia, M. C., Aguglia, U., . . . Gambardella, A. (2012). Neuroanatomic correlates of psychogenic nonepileptic seizures: A cortical thickness and VBM study. *Epilepsia*, 53(2), 377-385. doi:http://dx.doi.org/10.1111/j.1528-1167.2011.03347.x

Lacey, C., Cook, M., & Salzberg, M. (2007). The neurologist, psychogenic nonepileptic seizures, and borderline personality disorder. *Epilepsy & Behavior*, 11(4), 492-498. doi:10.1016/j.yebeh.2007.09.010



LaFrance Jr, W. C. (2010). Psychogenic nonepileptic "seizures" or "attacks"? It's not just semantics: Seizures. *Neurology*, 75(1), 87-88.

LaFrance Jr, W. C., Alosco, M. L., Davis, J. D., Tremont, G., Ryan, C. E., Keitner, G. I., . . . Blum, A. S. (2011). Impact of family functioning on quality of life in patients with psychogenic nonepileptic seizures versus epilepsy. *Epilepsia*, 52(2), 292-300.  
doi:10.1111/j.1528-1167.2010.02765.x

LaFrance Jr, W. C., Baird, G. L., Barry, J. J., Blum, A. S., Webb, A. F., Keitner, G. I., . . . Szaflarski, J. P. (2014). Multicenter pilot treatment trial for psychogenic nonepileptic seizures: A randomized clinical trial. *JAMA Psychiatry*, 71(9), 997-1005.

LaFrance Jr, W.C., Baker, G. A., Duncan, R., Goldstein, L. H., & Reuber, M. (2013). Minimum requirements for the diagnosis of psychogenic nonepileptic seizures: A staged approach: A report from the international league against epilepsy nonepileptic seizures task force. *Epilepsia*, 54(11), 2005-2018. doi:http://dx.doi.org/10.1111/epi.12356

LaFrance Jr, W. C., & Benbadis, S. R. (2011). Differentiating frontal lobe epilepsy from psychogenic nonepileptic seizures. *Neurologic Clinics*, 29(1), 149-162.  
doi:http://dx.doi.org/10.1016/j.ncl.2010.10.005

LaFrance Jr, W. C., DeLuca, M., Machan, J. T., & Fava, J. L. (2013). Traumatic brain injury and psychogenic nonepileptic seizures yield worse outcomes. *Epilepsia*, 54(4), 718-725. doi:10.1111/epi.12053

LaFrance Jr, W. C., de Marinis, A. J., Frank Webb, A., Machan, J. T., Rusch, M. D., & Kanner, A. M. (2012). Comparing standard medical care for nonepileptic seizures in Chile and the United States. *Epilepsy & Behavior*, 25(2), 224-229.  
doi:http://dx.doi.org/10.1016/j.yebeh.2012.07.028

LaFrance Jr, W. C., & Devinsky, O. (2002). Treatment of nonepileptic seizures. *Epilepsy & Behavior*, 3(5, Supplement), 19-23. doi:http://dx.doi.org/10.1016/S1525-5069(02)00505-4

LaFrance Jr, W. C., & Devinsky, O. (2004). The treatment of nonepileptic seizures: Historical perspectives and future directions. *Epilepsia*, 45, 15-21. doi:10.1111/j.0013-9580.2004.452002.x

LaFrance Jr, W. C., Keitner, G. I., Papandonatos, G. D., Blum, A. S., Machan, J. T., Ryan, C. E., & Miller, I. W. (2010). Pilot pharmacologic randomized controlled trial for psychogenic nonepileptic seizures. *Neurology*, 75(13), 1166-1173.  
doi:http://dx.doi.org/10.1212/WNL.0b013e3181f4d5a9

LaFrance Jr, W. C., Miller, I. W., Ryan, C. E., Blum, A. S., Solomon, D. A., Kelley, J. E., & Keitner, G. I. (2009). Cognitive behavioral therapy for psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 14(4), 591-596.  
doi:http://dx.doi.org/10.1016/j.yebeh.2009.02.016

LaFrance, Jr, W.C., & Plioplys, S. (2012). Neuropsychiatric disorders: Does semiology of psychogenic nonepileptic seizures matter? *Nature Reviews Neurology*, 8(6), 302-303.  
doi:http://dx.doi.org/10.1038/nrneurol.2012.79

LaFrance Jr, W. C., Reuber, M., & Goldstein, L. H. (2013). Management of psychogenic nonepileptic seizures. *Epilepsia*, 54, 53-67. doi:10.1111/epi.12106

LaFrance Jr, W. C., Rusch, M. D., & Machan, J. T. (2008). What is "treatment as usual" for nonepileptic seizures? *Epilepsy & Behavior*, 12(3), 388-394.  
doi:http://dx.doi.org/10.1016/j.yebeh.2007.12.017

LaFrance Jr, W. C., & Schachter, S. C. (2010). Historical approaches to treatments for psychogenic nonepileptic seizures. In S. C. Schachter, & W. C. LaFrance Jr (Eds.), *Gates and Rowan's nonepileptic seizures* (3rd ed., pp. 237-246). Cambridge: Cambridge University Press.

LaFrance Jr, W. C., & Syc, S. (2009). Depression and symptoms affect quality of life in psychogenic nonepileptic seizures. *Neurology*, 73(5), 366-371.  
doi:10.1212/WNL.0b013e3181b04c83; 10.1212/WNL.0b013e3181b04c83

LaFrance Jr, W.C., & Zimmerman, M. (2010). Classification of nonepileptic seizures. In Schachter, S.C. & LaFrance Jr, W.C. (Ed.), *Gates and Rowan's nonepileptic seizures* (3rd ed., pp. 199-212). Cambridge: Cambridge University Press.

Lally, N., Spence, W., McCusker, C., Craig, J., & Morrow, J. (2010). Psychological processes and histories associated with nonepileptic versus epileptic seizure presentations. *Epilepsy & Behavior*, 17(3), 360-365. doi:10.1016/j.yebeh.2009.12.001

Lancman, M. E., Asconape, J. J., Craven, W. J., Howard, G., & Penry, J. K. (1994). Predictive value of induction of psychogenic seizures by suggestion. *Annals of Neurology*, 35(3), 359-361. doi:10.1002/ana.410350319

Lancman, M. E., Asconape, J. J., Graves, S., & Gibson, P. A. (1994). Psychogenic seizures in children: Long-term analysis of 43 cases. *Journal of Child Neurology*, 9(4), 404-407.

Lancman, M. E., Brotherton, T. A., Asconape, J. J., & Penry, J. K. (1993). Psychogenic seizures in adults: A longitudinal analysis. *Seizure*, 2(4), 281-286.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (2005). *International Affective Picture System (IAPS): Instruction manual and affective ratings*. Gainesville, Florida: University of Florida.

Lanius, R. A., Vermetten, E., Loewenstein, R. J., Brand, B., Schmahl, C., Bremner, J. D., & Spiegel, D. (2010). Emotion modulation in PTSD: Clinical and neurobiological evidence for a dissociative subtype. *The American Journal of Psychiatry*, 167(6), 640-647. doi:10.1176/appi.ajp.2009.09081168 [doi]

Larsen, R. J., & Diener, E. (1987). Affect intensity as an individual difference characteristic: A review. *Journal of Research in Personality*, 21(1), 1-39.

Lawton, G., Baker, G. A., & Brown, R. J. (2008). Comparison of two types of dissociation in epileptic and nonepileptic seizures. *Epilepsy & Behavior*, 13(2), 333-336. doi:http://dx.doi.org/10.1016/j.yebeh.2008.04.015

Lawton, G., Mayor, R. J., Howlett, S., & Reuber, M. (2009). Psychogenic nonepileptic seizures and health-related quality of life: The relationship with psychological distress and other physical symptoms. *Epilepsy and Behavior*, 14(1), 167-171.  
doi:<http://dx.doi.org/10.1016/j.yebeh.2008.09.029>

Lazarus, J. P., Bhatia, M., Shukla, G., Padma, M. V., Tripathi, M., Shrivastava, A. K., . . . Jain, S. (2003). A study of nonepileptic seizures in an Indian population. *Epilepsy & Behavior*, 4(5), 496-499. doi:10.1016/S1525-5050(03)00118-5

Lazarus, R. S., & Folkman, S. (1987). Transactional theory and research on emotions and coping. *European Journal of Personality*, 1(3), 141-169.

Lee, T., Lim, S., Lee, K., Kim, H., & Choi, J. (2009). Conditioning-induced attentional bias for face stimuli measured with the emotional Stroop task. *Emotion*, 9(1), 134.

Leeman, B. A. (2009). Provocative techniques should not be used for the diagnosis of psychogenic nonepileptic seizures. *Epilepsy and Behavior*, 15(2), 110-114.  
doi:<http://dx.doi.org/10.1016/j.yebeh.2009.01.013>

Lelliott, P., & Fenwick, P. (1991). Cerebral pathology in pseudoseizures. *Acta Neurologica Scandinavica*, 83(2), 129-132.

Lempert, T., & Schmidt, D. (1990). Natural history and outcome of psychogenic seizures: A clinical study in 50 patients. *Journal of Neurology*, 237(1), 35-38.

Leppänen, J. M., Milders, M., Bell, J. S., Terriere, E., & Hietanen, J. K. (2004). Depression biases the recognition of emotionally neutral faces. *Psychiatry Research*, 128(2), 123-133.

Lesser, R. P. (1996). Psychogenic seizures. *Neurology*, 46(6), 1499-1507.

Lesser, R. P. (2002). Nonepileptic seizures: Psychiatric aspects. *Epilepsy & Behavior*, 3(1), 34-38. doi:10.1006/ebbeh.2001.0309

Lesser, R. P. (2003). Treating psychogenic nonepileptic seizures: Easier said than done. *Annals of Neurology*, 53(3), 285-286. doi:10.1002/ana.10542

Levine, D., Marziali, E., & Hood, J. (1997). Emotion processing in borderline personality disorders. *The Journal of Nervous and Mental Disease*, 185(4), 240-246.

Levy, K. J. (1980). A Monte Carlo study of analysis of covariance under violations of the assumptions of normality and equal regression slopes. *Educational and Psychological Measurement*, 40(4), 835-840.

Li, R., Liu, K., Ma, X., Li, Z., Duan, X., An, D., . . . Chen, H. (2014). Altered functional connectivity patterns of the insular subregions in psychogenic nonepileptic seizures. *Brain Topography*, doi:10.1007/s10548-014-0413-3 [doi]

Lichter, I., Goldstein, L. H., Toone, B. K., & Mellers, J. D. C. (2004). Nonepileptic seizures following general anesthetics: A report of five cases. *Epilepsy & Behavior*, 5(6), 1005-1013. doi:10.1016/j.yebeh.2004.09.003

Lim, S., & Kim, J. (2005). Cognitive processing of emotional information in depression, panic, and somatoform disorder. *Journal of Abnormal Psychology*, 114(1), 50.

Litwin, R., & Cardena, E. (2001). Demographic and seizure variables, but not hypnotizability or dissociation, differentiated psychogenic from organic seizures. *Journal of Trauma & Dissociation*, 1(4), 99-122.

Livesley, W. J., & Jackson, D. N. (2002). *Manual for the Dimensional Assessment of Personality Pathology*. Port Huron: Sigma Press.

Locke, D. E. C., Berry, D. T. R., Fakhoury, T. A., & Schmitt, F. A. (2006). Relationship of indicators of neuropathology, psychopathology, and effort to neuropsychological results in patients with epilepsy or psychogenic non-epileptic seizures. *Journal of Clinical and Experimental Neuropsychology*, 28(3), 325-340. doi:10.1080/13803390490918183

Lykken, D. T., & Venables, P. H. (1971). Direct measurement of skin conductance: A proposal for standardization. *Psychophysiology*, 8(5), 656-672.

Lynch, T. R., Rosenthal, M. Z., Kosson, D. S., Cheavens, J. S., Lejuez, C., & Blair, R. (2006). Heightened sensitivity to facial expressions of emotion in borderline personality disorder. *Emotion*, 6(4), 647.

MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology*, 95(1), 15.

Magaudda, A., Gugliotta, S. C., Tallarico, R., Buccheri, T., Alfa, R., & Lagana, A. (2011). Identification of three distinct groups of patients with both epilepsy and psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 22(2), 318-323.  
doi:10.1016/j.yebeh.2011.07.005

Marchetti, R. L., Kurcgant, D., Gallucci Neto, J., von Bismark, M. A., & Fiore, L. A. (2009). Evaluating patients with suspected nonepileptic psychogenic seizures. *Journal of Neuropsychiatry and Clinical Neurosciences*, 21(3), 292-298.

Marchetti, R. L., Kurcgant, D., Neto, J. G., von Bismark, M. A., Marchetti, L. B., & Fiore, L. A. (2008). Psychiatric diagnoses of patients with psychogenic non-epileptic seizures. *Seizure-European Journal of Epilepsy*, 17(3), 247-253. doi:10.1016/j.seizure.2007.07.006

Mari, F., Di Bonaventura, C., Vanacore, N., Fattouch, J., Vaudano, A. E., Egeo, G., . . . Giallonardo, A. T. (2006). Video-EEG study of psychogenic nonepileptic seizures: Differential characteristics in patients with and without epilepsy. *Epilepsia*, 47, 64-67.  
doi:10.1111/j.1528-1167.2006.00880.x

Marissen, M. A., Meuleman, L., & Franken, I. H. (2010). Altered emotional information processing in borderline personality disorder: An electrophysiological study. *Psychiatry Research: Neuroimaging*, 181(3), 226-232.

Markoula, S., De Tisi, J., Foong, J., & Duncan, J. S. (2013). Psychogenic nonepileptic seizures after adult epilepsy surgery. *Epilepsy Currents*, 13, 440.

Marmar, C. R., Weiss, D. S., & Metzler, T. J. (1997). The Peritraumatic Dissociative Experiences Questionnaire. In J. P. Wilson, & T. M. Keane (Eds.), *Assessing psychological trauma and PTSD* (pp. 412-428). New York: Guildford Press.

Marmar, C. R., Weiss, D. S., & Metzler, T. J. (1998). Peritraumatic dissociation and posttraumatic stress disorder. *Trauma, memory and dissociation* (pp. 229-252). Washington: American Psychiatric Press.

Marquez, A. V., Farias, S. T., Apperson, M., Koopmans, S., Jorgensen, J., Shatzel, A., & Alsaadi, T. M. (2004). Psychogenic nonepileptic seizures are associated with an increased risk of obesity. *Epilepsy & Behavior*, 5(1), 88-93.

Martin, R., Burneo, J. G., Prasad, A., Powell, T., Faught, E., Knowlton, R., . . . Kuzniecky, R. (2003). Frequency of epilepsy in patients with psychogenic seizures monitored by video-EEG. *Neurology*, 61(12), 1791-1792.

Martin, R. C., Gilliam, F. G., Kilgore, M., Faught, E., & Kuzniecky, R. (1998). Improved health care resource utilization following video-EEG-confirmed diagnosis of nonepileptic psychogenic seizures. *Seizure*, 7(5), 385-390. doi:10.1016/S1059-1311(05)80007-X

Martinez-Taboas, A. (2002). The role of hypnosis in the detection of psychogenic seizures. *American Journal of Clinical Hypnosis*, 45(1), 11-20.

Martinez-Taboas, A., Lewis-Fernandez, R., Sar, V., & Agarwal, A. L. (2010). Cultural aspects of psychogenic nonepileptic seizures. In S.C. Schachter, & W.C. LaFrance Jr (Eds.), *Gates and Rowan's nonepileptic seizures* (3rd ed., pp. 121-130). Cambridge: Cambridge University Press.

Matsumoto, D., Keltner, D., Shiota, M. N., O'Sullivan, M., & Frank, M. (2008). Facial expressions of emotion. In M. Lewis, J. M. Haviland-Jones & L. Feldman Barrett (Eds.), *Handbook of emotions*. (3rd ed., pp. 211-234). New York: Guilford Publications.

Maxwell, J., & Davidson, R. (2004). Unequally masked: Indexing differences in the perceptual salience of "unseen" facial expressions. *Cognition & Emotion*, 18(8), 1009-1026.

Mayor, R., Brown, R. J., Cock, H., House, A., Howlett, S., Singhal, S., . . . Reuber, M. (2012). Short-term outcome of psychogenic non-epileptic seizures after communication of the diagnosis. *Epilepsy & Behavior*, 25(4), 676-681. doi:10.1016/j.yebeh.2012.09.033

Mayor, R., Brown, R. J., Cock, H., House, A., Howlett, S., Smith, P., & Reuber, M. (2013). A feasibility study of a brief psycho-educational intervention for psychogenic nonepileptic seizures. *Seizure*, 22(9), 760-765.  
doi:http://dx.doi.org/10.1016/j.seizure.2013.06.008

Mazza, M., Della Marca, G., Martini, A., Scoppetta, M., Vollono, C., Valenti, M. A., . . . Mazza, S. (2009). Non-epileptic seizures (NES) are predicted by depressive and dissociative symptoms. *Epilepsy Research*, 84(2-3), 91-96.  
doi:10.1016/j.eplepsyres.2008.12.008

McDade, G., & Brown, S. W. (1992). Non-epileptic seizures: Management and predictive factors of outcome. *Seizure*, 1(1), 7-10. doi:10.1016/1059-1311(92)90047-5

McGonigal, A., Oto, M., Russell, A. J. C., Greene, J., & Duncan, R. (2002). Outpatient video EEG recording in the diagnosis of non-epileptic seizures: A randomised controlled trial of simple suggestion techniques. *Journal of Neurology Neurosurgery and Psychiatry*, 72(4), 549-551.

McKenzie, P., Oto, M., Russell, A., Pelosi, A., & Duncan, R. (2010). Early outcomes and predictors in 260 patients with psychogenic nonepileptic attacks. *Neurology*, 74(1), 64-69. doi:10.1212/WNL.0b013e3181c7da6a

McLean, T., & Dyer, C. (2003). Treatment of psychogenic pseudoseizures in an adolescent with a history of epilepsy. *Clinical Psychologist*, 7(2), 109-120.



McMillan, K. K., Pugh, M. J., Hamid, H., Salinsky, M., Pugh, J., Noel, P. H., . . . LaFrance, W. C., Jr. (2014). Providers' perspectives on treating psychogenic nonepileptic seizures: Frustration and hope. *Epilepsy & Behavior*, 37, 276-281. doi:10.1016/j.yebeh.2014.07.001

McNally, K. A., Schefft, B. K., Szaflarski, J. P., Howe, S. R., Yeh, H., & Privitera, M. D. (2009). Application of signal detection theory to verbal memory testing to distinguish patients with psychogenic nonepileptic seizures from patients with epileptic seizures. *Epilepsy & Behavior*, 14(4), 597-603. doi:10.1016/j.yebeh.2009.01.012

McNally, R. J., English, G. E., & Lipke, H. J. (1993). Assessment of intrusive cognition in PTSD: Use of the modified stroop paradigm. *Journal of Traumatic Stress*, 6(1), 33-41.

Meador, K. J., Baker, G. A., Browning, N., Clayton-Smith, J., Combs-Cantrell, D. T., Cohen, M., . . . Pennell, P. B. (2009). Cognitive function at 3 years of age after fetal exposure to antiepileptic drugs. *New England Journal of Medicine*, 360(16), 1597-1605.

Meletti, S., Benuzzi, F., Cantalupo, G., Rubboli, G., Tassinari, C. A., & Nichelli, P. (2009). Facial emotion recognition impairment in chronic temporal lobe epilepsy. *Epilepsia*, 50(6), 1547-1559.

Meletti, S., Benuzzi, F., Rubboli, G., Cantalupo, G., Stanzani Maserati, M., Nichelli, P., & Tassinari, C. A. (2003). Impaired facial emotion recognition in early-onset right mesial temporal lobe epilepsy. *Neurology*, 60(3), 426-431.

Mellers, J. D. C. (2005). The approach to patients with "non-epileptic seizures". *Postgraduate Medical Journal*, 81(958), 498-504. doi:10.1136/pgmj.2004.029785

Mellers, J. D. C. (2009). The diagnosis and management of dissociative seizures. In J.W. Sander, M.C. Walker, & J.E. Smalls (Eds.), *From benchside to bedside. A practical guide to epilepsy*. (Chapter 19). London, UK: ILAE and the National Society for Epilepsy.

Merens, W., Van der Does, A.J. Willem, & Spinhoven, P. (2007). The effects of serotonin manipulations on emotional information processing and mood. *Journal of Affective Disorders*, 103(1), 43-62.

Merikle, P. (2007; 2007). Preconscious processing. In M. Velmans, & S. Schneider (Eds.), *The Blackwell Companion to Consciousness* (pp. 512-524). Oxford: Blackwell Publishing Ltd.

Merkl, A., Ammelburg, N., Aust, S., Roepke, S., Reinecker, H., Trahms, L., . . . Sander, T. (2010). Processing of visual stimuli in borderline personality disorder: A combined behavioural and magnetoencephalographic study. *International Journal of Psychophysiology*, 78(3), 257-264.

Metin, S. Z., Ozmen, M., Metin, B., Talasman, S., Yeni, S. N., & Ozkara, C. (2013). Treatment with group psychotherapy for chronic psychogenic nonepileptic seizures. *Epilepsy and Behavior*, 28(1), 91-94. doi:<http://dx.doi.org/10.1016/j.yebeh.2013.03.023>

Metrick, M. E., Ritter, F. J., Gates, J. R., Jacobs, M. P., Skare, S. S., & Loewenson, R. B. (1991). Nonepileptic events in childhood. *Epilepsia*, 32(3), 322-328.

Mihaescu, M., & Malow, B. A. (2003). Sleep disorders: A sometimes forgotten cause of nonepileptic spells. *Epilepsy & Behavior*, 4(6), 784-787.  
doi:<http://dx.doi.org/10.1016/j.yebeh.2003.08.024>

Miller, H. R. (1983). Psychogenic seizures treated by hypnosis. *The American Journal of Clinical Hypnosis*, 25(4), 248-252.

Miller, G. A., & Chapman, J. P. (2001). Misunderstanding analysis of covariance. *Journal of Abnormal Psychology*, 110(1), 40.

Millon, T. (1992). Millon Multiaxial Clinical Inventory: I & II. *Journal of Counseling & Development*, 70(3), 421-426. doi: 10.1002/j.1556-6676.1992.tb01627.x

Minzenberg, M. J., Poole, J. H., & Vinogradov, S. (2006). Social-emotion recognition in borderline personality disorder. *Comprehensive Psychiatry*, 47(6), 468-474.

Mitchell, J. W., Ali, F., & Cavanna, A. E. (2012). Dissociative experiences and quality of life in patients with non-epileptic attack disorder. *Epilepsy & Behavior*, 25(3), 307-312. doi:10.1016/j.yebeh.2012.08.022

Modestin, J., Ebner, G., Junghan, M., & Erni, T. (1996). Dissociative experiences and dissociative disorders in acute psychiatric inpatients. *Comprehensive Psychiatry*, 37(5), 355-361. doi:S0010-440X(96)90017-6 [pii]

Moene, F. C., Spinhoven, P., Hoogduin, K. A., & van Dyck, R. (2002). A randomised controlled clinical trial on the additional effect of hypnosis in a comprehensive treatment programme for in-patients with conversion disorder of the motor type. *Psychotherapy and Psychosomatics*, 71 (2), 66-76.

Mogg, K., & Bradley, B. P. (1999). Orienting of attention to threatening facial expressions presented under conditions of restricted awareness. *Cognition & Emotion*, 13(6), 713-740.

Mogg, K., & Bradley, B. P. (2002). Selective orienting of attention to masked threat faces in social anxiety. *Behaviour Research and Therapy*, 40(12), 1403-1414.

Mökleby, K., Blomhoff, S., Malt, U. F., Dahlstrom, A., Tauboll, E., & Gjerstad, L. (2002). Psychiatric comorbidity and hostility in patients with psychogenic nonepileptic seizures compared with somatoform disorders and healthy controls. *Epilepsia*, 43(2), 193-198.

Montagne, B., Sierra, M., Medford, N., Hunter, E., Baker, D., Kessels, R. P., . . . David, A. S. (2007). Emotional memory and perception of emotional faces in patients suffering from depersonalization disorder. *British Journal of Psychology*, 98(3), 517-527.

Moore, D. B., Abou-Khalil, B., Fakhoury, T., & Mathews, G. (1998). Preictal behavior in psychogenic and epileptic seizures. *Journal of Epilepsy*, 11(4), 182-186. doi:http://dx.doi.org/10.1016/S0896-6974(98)00011-5

Moore, P. M., & Baker, G. A. (1997). Non-epileptic attack disorder: A psychological perspective. *Seizure*, 6(6), 429-434. doi:10.1016/S1059-1311(97)80016-7

Moore, P. M., Baker, G. A., Mcdade, G., Chadwick, D., & Brown, S. (1994). Epilepsy, pseudoseizures and perceived family characteristics - a controlled-study. *Epilepsy Research*, 18(1), 75-83. doi:10.1016/0920-1211(94)90035-3

Moos, B. S., & Moos, R. H. (1981). *The Family Environment Scale (FES)*. Menlo Park: CA: Mind Garden Inc.

Morey, L. C. (1991). *Personality Assessment Inventory (PAI)*. Psychological Assessment Resources (PAR).

Morrison, I., & Razvi, S. S. M. (2011). Driving regulations and psychogenic non-epileptic seizures: Perspectives from the United Kingdom. *Seizure*, 20(2), 177-180. doi:10.1016/j.seizure.2010.11.011

Mostacci, B., Bisulli, F., Alvisi, L., Licchetta, L., Baruzzi, A., & Tinuper, P. (2011). Ictal characteristics of psychogenic nonepileptic seizures: What we have learned from video/EEG recordings-A literature review. *Epilepsy and Behavior*, 22(2), 144-153. doi:http://dx.doi.org/10.1016/j.yebeh.2011.07.003

Mueller, J., Alpers, G. W., & Reim, N. (2006). Dissociation of rated emotional valence and stroop interference in observer-rated alexithymia. *Journal of Psychosomatic Research*, 61(2), 261-269.

Mulder, R. T., Beautrais, A. L., Joyce, P. R., & Fergusson, D. M. (1998). Relationship between dissociation, childhood sexual abuse, childhood physical abuse, and mental illness in a general population sample. *The American Journal of Psychiatry*, 155(6), 806-811.

Muller, T., Merschhemke, M., Dehnicke, C., Sanders, M., & Meencke, H. J. (2002). Improving diagnostic procedure and treatment in patients with non-epileptic seizures (NES). *Seizure*, 11(2), 85-89. doi:10.1053/seiz.2002.0624

Mungen, B., Berilgen, M. S., & Arikanoglu, A. (2010). Autonomic nervous system functions in interictal and postictal periods of nonepileptic psychogenic seizures and its comparison with epileptic seizures. *Seizure*, 19(5), 269-273.

doi:<http://dx.doi.org/10.1016/j.seizure.2010.04.002>

Myers, L., Fleming, M., Lancman, M., Perrine, K., & Lancman, M. (2013). Stress coping strategies in patients with psychogenic non-epileptic seizures and how they relate to trauma symptoms, alexithymia, anger and mood. *Seizure*, 22(8), 634-639.

doi:<http://dx.doi.org/10.1016/j.seizure.2013.04.018>

Myers, L., Laban-Grant, O., Matzner, B., & Lancman, M. (2012). Psychogenic non-epileptic seizures: Predisposing factors to diminished quality of life. *Epilepsy & Behavior*, 25(3), 358-362. doi:<http://dx.doi.org/10.1016/j.yebeh.2012.08.024>

Myers, L., Perrine, K., Fleming, M., & Lancman, M. (2013). Psychological trauma in patients with psychogenic nonepileptic seizures: Trauma characteristics and those who develop PTSD. *Epilepsy & Behavior*, 28(1), 121-126.

doi:<http://dx.doi.org/10.1016/j.yebeh.2013.03.033>

Myers, L., Zeng, R., Perrine, K., Lancman, M., & Lancman, M. (2014). Cognitive differences between patients who have psychogenic nonepileptic seizures (PNESs) and posttraumatic stress disorder (PTSD) and patients who have PNESs without PTSD. *Epilepsy & Behavior*, 37(0), 82-86. doi:<http://dx.doi.org/10.1016/j.yebeh.2014.06.009>

Mykletun, A., Stordal, E., & Dahl, A. A. (2001). Hospital Anxiety and Depression (HAD) Scale: Factor structure, item analyses and internal consistency in a large population. *The British Journal of Psychiatry*, 179, 540-544.

Nadkarni, S., & Devinsky, O. (2005). Psychotropic effects of antiepileptic drugs. *Epilepsy Currents*, 5(5), 176-181.

Najström, M., & Jansson, B. (2007). Skin conductance responses as predictor of emotional responses to stressful life events. *Behaviour Research and Therapy*, 45(10), 2456-2463.

Nashiro, K., Sakaki, M., & Mather, M. (2012). Age differences in brain activity during emotion processing: Reflections of age-related decline or increased emotion regulation? *Gerontology*, 58(2), 156-163. doi:10.1159/000328465 [doi]

Ney, G. C., Barr, W. B., Napolitano, C., Decker, R., & Schaul, N. (1998). New-onset psychogenic seizures after surgery for epilepsy. *Archives of Neurology*, 55(5), 726-730. doi:10.1001/archneur.55.5.726

Ney, G. C., Lantos, G., Barr, W. B., & Schaul, N. (1994). Cerebellar atrophy in patients with long-term phenytoin exposure and epilepsy. *Archives of Neurology*, 51(8), 767-771.

Ney, G. C., Zimmerman, C., & Schaul, N. (1996). Psychogenic status epilepticus induced by a provocative technique. *Neurology*, 46(2), 546-547.

Nicholson, T. R. J., & Kanaan, R. A. A. (2009). Conversion disorder. *Psychiatry*, 8(5), 164-169. doi:http://dx.doi.org/10.1016/j.mppsy.2009.03.001

Nicholson, T. R. J., Stone, J., & Kanaan, R. A. A. (2011). Conversion disorder: A problematic diagnosis. *Journal of Neurology Neurosurgery and Psychiatry*, 82(11), 1267-1273. doi:10.1136/jnnp.2008.171306

Nicol, K., Pope, M., & Hall, J. (2014). Facial emotion recognition in borderline personality: An association, with childhood experience. *Psychiatry Research*, 218(1), 256-258.

Niedermaier, O. N., Smith, M. L., Beightol, L. A., Zukowska-Grojec, Z., Goldstein, D. S., & Eckberg, D. L. (1993). Influence of cigarette smoking on human autonomic function. *Circulation*, 88(2), 562-571.

Nijenhuis, E. R. (2001). Somatoform dissociation: Major symptoms of dissociative disorders. *Journal of Trauma & Dissociation*, 1(4), 7-32.

Nijenhuis, E. R., Spinhoven, P., van Dyck, R., van Der Hart, O., & Vanderlinden, J. (1996). The development and psychometric characteristics of the Somatoform Dissociation Questionnaire (SDQ-20). *The Journal of Nervous and Mental Disease*, 184(11), 688-694.

Nijenhuis, E. R., Spinhoven, P., van Dyck, R., van der Hart, O., & Vanderlinden, J. (1997). The development of the Somatoform Dissociation Questionnaire (SDQ-5) as a screening instrument for dissociative disorders. *Acta Psychiatrica Scandinavica*, 96(5), 311-318.

Nijenhuis, E. R., Spinhoven, P., van Dyck, R., van der Hart, O., & Vanderlinden, J. (1998). Degree of somatoform and psychological dissociation in dissociative disorder is correlated with reported trauma. *Journal of Traumatic Stress*, 11(4), 711-730. doi:10.1023/A:1024493332751 [doi]

Nijenhuis, E. R., van der Hart, O., & Vanderlinden, J. (1999). The Traumatic Experiences Checklist (TEC). In E. R. Nijenhuis (Ed.), *Somatoform dissociation: Phenomena, measurement, and theoretical issues* (pp. 223-229). Assen, Netherlands: Von Gorcum.

Nijenhuis, E. R., van Dyck, R., ter Kuile, M. M., Mourits, M. J., Spinhoven, P., & van der Hart, O. (2003). Evidence for associations among somatoform dissociation, psychological dissociation and reported trauma in patients with chronic pelvic pain. *Journal of Psychosomatic Obstetrics and Gynaecology*, 24(2), 87-98.

Nijenhuis, E. R., Vanderlinden, J., & Spinhoven, P. (1998). Animal defensive reactions as a model for trauma-induced dissociative reactions. *Journal of Traumatic Stress*, 11(2), 243-260. doi:10.1023/A:1024447003022 [doi]

Nowak, D. A., & Fink, G. R. (2009). Psychogenic movement disorders: Aetiology, phenomenology, neuroanatomical correlates and therapeutic approaches. *Neuroimage*, 47(3), 1015-1025. doi:<http://dx.doi.org/10.1016/j.neuroimage.2009.04.082>

Oakley, D. A. (1999). Hypnosis and conversion hysteria: A unifying model. *Cognitive Neuropsychiatry*, 4(3), 243-265.

Oathes, D. J., & Ray, W. J. (2008). Dissociative tendencies and facilitated emotional processing. *Emotion*, 8(5), 653-661. doi:10.1037/a0013442 [doi]

O'Brien, F. M., Fortune, G. M., Dicker, P., O'Hanlon, E., Cassidy, E., Delanty, N., . . . Murphy, K. C. (2015). Psychiatric and neuropsychological profiles of people with psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 43(0), 39-45. doi:<http://dx.doi.org/10.1016/j.yebeh.2014.11.012>

Office for National Statistics (ONS). (2010). SOC 2010 volume 3: The national statistics socio-economic classification (NS-SEC). UK: Office for National Statistics. Retrieved from: <http://www.ons.gov.uk/ons/guide-method/classifications/current-standard-classifications/soc2010/soc2010-volume-3-ns-sec--rebased-on-soc2010--user-manual/index.html>

Office for National Statistics (ONS). (2013). *Focus on: Violent crime and sexual offences, 2011/2012*. (). UK: Office for National Statistics.

O'Hanlon, S., Liston, R., & Delanty, N. (2012). Psychogenic nonepileptic seizures: Time to abandon the term pseudoseizures. *Archives of Neurology*, 69(10), 1349-1350. doi:<http://dx.doi.org/10.1001/archneurol.2012.829>

Öhman, A. (2002). Automaticity and the amygdala: Nonconscious responses to emotional faces. *Current Directions in Psychological Science*, 11(2), 62-66.

Öhman, A., Flykt, A., & Esteves, F. (2001). Emotion drives attention: Detecting the snake in the grass. *Journal of Experimental Psychology: General*, 130(3), 466.



Öhman, A., Flykt, A., & Lundqvist, D. (2000). Unconscious emotion: Evolutionary perspectives, psychophysiological data, and neuropsychological mechanisms. In R. Lane, & L. Nadel (Eds.), *The cognitive neuroscience of emotion* (pp. 296-327). New York: Oxford University Press.

Opherk, C., & Hirsch, L. J. (2002). Ictal heart rate differentiates epileptic from non-epileptic seizures. *Neurology*, 58(4), 636-638.

O'Sullivan, S. S., Spillane, J. E., McMahon, E. M., Sweeney, B. J., Galvin, R. J., McNamara, B., & Cassidy, E. M. (2007). Clinical characteristics and outcome of patients diagnosed with psychogenic nonepileptic seizures: A 5-year review. *Epilepsy & Behavior*, 11(1), 77-84. doi:10.1016/j.yebeh.2007.04.003

Oto, M., & Reuber, M. (2014). Psychogenic non-epileptic seizures: Aetiology, diagnosis and management. *Advances in Psychiatric Treatment*, 20(1), 13-22.  
doi:http://dx.doi.org/10.1192/apt.bp.113.011171

Owczarek, K., & Jędrzejczak, J. (2001). Patients with coexistent psychogenic pseudoepileptic and epileptic seizures: A psychological profile. *Seizure*, 10(8), 566-569.  
doi:http://dx.doi.org/10.1053/seiz.2001.0556

Ozcetin, A., Belli, H., Ertem, U., Bahcebasi, T., Ataoglu, A., & Canan, F. (2009). Childhood trauma and dissociation in women with pseudoseizure-type conversion disorder. *Nordic Journal of Psychiatry*, 63(6), 462-468. doi:10.3109/08039480903029728

Ozer, E. J., Best, S. R., Lipsey, T. L., & Weiss, D. S. (2003). Predictors of posttraumatic stress disorder and symptoms in adults: A meta-analysis. *Psychological Bulletin*, 129(1), 52-73.

Pakalnis, A., & Paolicchi, J. (2000). Psychogenic seizures after head injury in children. *Journal of Child Neurology*, 15(2), 78-80. doi:10.1177/088307380001500202

Pakalnis, A., & Paolicchi, J. (2003). Frequency of secondary conversion symptoms in children with psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 4(6), 753-756.

Pakalnis, A., Paolicchi, J., & Gilles, E. (2000). Psychogenic status epilepticus in children: Psychiatric and other risk factors. *Neurology*, 54(4), 969-970.

Parain, D., & Chastan, N. (2014). Large-field repetitive transcranial magnetic stimulation with circular coil in the treatment of functional neurological symptoms. *Neurophysiologie Clinique/Clinical Neurophysiology*, 44(4), 425-431.  
doi:<http://dx.doi.org/10.1016/j.neucli.2014.04.004>

Park, J. H., Bokma, J., Chapple, K., & Caplan, J. P. (2014). A retrospective study of polyallergy as a marker of nonepileptic seizures in the epilepsy monitoring unit. *Psychosomatics*, 55(6), 566-571. doi:<http://dx.doi.org/10.1016/j.psych.2014.05.004>

Parra, J., Iriarte, J., & Kanner, A. M. (1999). Are we overusing the diagnosis of psychogenic non-epileptic events? *Seizure*, 8(4), 223-227. doi:10.1053/seiz.1999.0285

Parra, J., Iriarte, J., Kanner, A. M., & Bergen, D. C. (1998). De novo psychogenic nonepileptic seizures after epilepsy surgery. *Epilepsia*, 39(5), 474-477.

Patel, H., Scott, E., Dunn, D., & Garg, B. (2007). Nonepileptic seizures in children. *Epilepsia*, 48(11), 2086-2092. doi:<http://dx.doi.org/10.1111/j.1528-1167.2007.01200.x>

Patidar, Y., Gupta, M., Khwaja, G., Chowdhury, D., Batra, A., & Dasgupta, A. (2013). Clinical profile of psychogenic non-epileptic seizures in adults: A study of 63 cases. *Annals of Indian Academy of Neurology*, 16(2), 157-162.  
doi:<http://dx.doi.org/10.4103/0972-2327.112451>

Pavlova, M. K., Allen, R. M., & Dworetzky, B. A. (2014). Sleep in psychogenic nonepileptic seizures and related disorders. *Clinical EEG and Neuroscience*,  
doi:1550059414560565 [pii]

Peckham, A. D., McHugh, R. K., & Otto, M. W. (2010). A meta-analysis of the magnitude of biased attention in depression. *Depression and Anxiety*, 27(12), 1135-1142.

Pedrosa Gil, F., Ridout, N., Kessler, H., Neuffer, M., Schoechlin, C., Traue, H. C., & Nickel, M. (2009). Facial emotion recognition and alexithymia in adults with somatoform disorders. *Depression and Anxiety*, 26(1), E26-E33.

Peduzzi, P., Concato, J., Kemper, E., Holford, T. R., & Feinstein, A. R. (1996). A simulation study of the number of events per variable in logistic regression analysis. *Journal of Clinical Epidemiology*, 49(12), 1373-1379.

Perez, D. L., Dworetzky, B. A., Dickerson, B. C., Leung, L., Cohn, R., Baslet, G., & Silbersweig, D. A. (2014). An integrative neurocircuit perspective on psychogenic nonepileptic seizures and functional movement disorders: Neural functional unawareness. *Clinical EEG and Neuroscience*, doi:1550059414555905 [pii]

Pessoa, L. (2005). To what extent are emotional visual stimuli processed without attention and awareness? *Current Opinion in Neurobiology*, 15(2), 188-196.

Petkar, S., Hamid, T., Iddon, P., Clifford, A., Rice, N., Claire, R., . . . Fitzpatrick, A. P. (2012). Prolonged implantable electrocardiographic monitoring indicates a high rate of misdiagnosis of epilepsy--REVISE study. *Europace : European Pacing, Arrhythmias, and Cardiac Electrophysiology : Journal of the Working Groups on Cardiac Pacing, Arrhythmias, and Cardiac Cellular Electrophysiology of the European Society of Cardiology*, 14(11), 1653-1660. doi:10.1093/europace/eus185; 10.1093/europace/eus185

Phillips, M. L., Drevets, W. C., Rauch, S. L., & Lane, R. (2003). Neurobiology of emotion perception II: Implications for major psychiatric disorders. *Biological Psychiatry*, 54(5), 515-528.

Pillai, J. A., & Haut, S. R. (2012). Patients with epilepsy and psychogenic non-epileptic seizures: An inpatient video-EEG monitoring study. *Seizure*, 21(1), 24-27. doi:10.1016/j.seizure.2011.09.002

Pinker, S. (1997). *How the mind works*. New York: Norton & Company.

- Pintor, L., Bailles, E., Matrai, S., Carreno, M., Donaire, A., Boget, T., . . . Bargallo, N. (2010). Efficiency of venlafaxine in patients with psychogenic nonepileptic seizures and anxiety and/or depressive disorders. *Journal of Neuropsychiatry and Clinical Neurosciences*, 22(4), 401-408. doi:<http://dx.doi.org/10.1176/appi.neuropsych.22.4.401>
- Plana, I., Lavoie, M., Battaglia, M., & Achim, A. M. (2014). A meta-analysis and scoping review of social cognition performance in social phobia, posttraumatic stress disorder and other anxiety disorders. *Journal of Anxiety Disorders*, 28(2), 169-177. doi:<http://dx.doi.org/10.1016/j.janxdis.2013.09.005>
- Plioplys, S., Doss, J., Siddarth, P., Bursch, B., Falcone, T., Forgey, M., . . . Caplan, R. (2014). A multisite controlled study of risk factors in pediatric psychogenic nonepileptic seizures. *Epilepsia*, 55(11), 1739-1747. doi:10.1111/epi.12773
- Plug, L., Sharrack, B., & Reuber, M. (2009). Seizure metaphors differ in patients' accounts of epileptic and psychogenic nonepileptic seizures. *Epilepsia*, 50(5), 994-1000. doi:10.1111/j.1528-1167.2008.01798.x; 10.1111/j.1528-1167.2008.01798.x
- Poljac, E., Montagne, B., & de Haan, E. H. (2011). Reduced recognition of fear and sadness in post-traumatic stress disorder. *Cortex*, 47(8), 974-980.
- Pollatos, O., Herbert, B. M., Wankner, S., Dietel, A., Wachsmuth, C., Henningsen, P., & Sack, M. (2011). Autonomic imbalance is associated with reduced facial recognition in somatoform disorders. *Journal of Psychosomatic Research*, 71(4), 232-239.
- Ponnusamy, A., Marques, J. L. B., & Reuber, M. (2011). Heart rate variability measures as biomarkers in patients with psychogenic nonepileptic seizures: Potential and limitations. *Epilepsy & Behavior*, 22(4), 685-691. doi:10.1016/j.yebeh.2011.08.020
- Ponnusamy, A., Marques, J. L. B., & Reuber, M. (2012). Comparison of heart rate variability parameters during complex partial seizures and psychogenic nonepileptic seizures. *Epilepsia*, 53(8), 1314-1321. doi:10.1111/j.1528-1167.2012.03518.x

- Portuguez, M. W., Da Costa, D. I., Marroni, S. P., Pagliarini, V., & Vieira, K. (2007). Neuropsychological aspects of psychogenic nonepileptic seizures. *Journal of Epilepsy and Clinical Neurophysiology*, 13(4 SUPPL 1), 24-27. doi:http://dx.doi.org/10.1590/S1676-26492007000500005
- Pouretamad, H. R., Thompson, P. J., & Fenwick, P. B. C. (1998). Impaired sensorimotor gating in patients with non-epileptic seizures. *Epilepsy Research*, 31(1), 1-12. doi:10.1016/S0920-1211(98)00014-X
- Prigatano, G. P., Amin, K., & Rosenstein, L. D. (1995). *Administration and scoring manual for the BNI screen for higher cerebral functions*. Phoenix: Barrow Neurological Institute.
- Prigatano, G. P., & Kirlin, K. A. (2009). Self-appraisal and objective assessment of cognitive and affective functioning in persons with epileptic and nonepileptic seizures. *Epilepsy & Behavior*, 14(2), 387-392. doi:http://dx.doi.org/10.1016/j.yebeh.2008.12.001
- Prigatano, G. P., Stonnington, C. M., & Fisher, R. S. (2002). Psychological factors in the genesis and management of nonepileptic seizures: Clinical observations. *Epilepsy & Behavior*, 3(4), 343-349. doi:10.1016/S1525-5050(02)00053-7
- Proença, I. C. G. F., Castro, L. H. M., Jorge, C. L., & Marchetti, R. L. (2011). Emotional trauma and abuse in patients with psychogenic nonepileptic seizures. *Epilepsy and Behavior*, 20(2), 331-333. doi:http://dx.doi.org/10.1016/j.yebeh.2010.11.015
- Prueter, C., Schultz-Venrath, U., & Rimpau, W. (2002). Dissociative and associated psychopathological symptoms in patients with epilepsy, pseudoseizures, and both seizure forms. *Epilepsia*, 43(2), 188-192. doi:10.1046/j.1528-1157.2002.45900.x
- Putman, P., Hermans, E., & Van Honk, J. (2004). Emotional Stroop performance for masked angry faces: It's BAS, not BIS. *Emotion*, 4(3), 305.
- Putnam, F. W. (1997). *Dissociation in children and adolescents: A developmental perspective*. New York: Guilford Press.

Quigg, M., Armstrong, R. F., Farace, E., & Fountain, N. B. (2002). Quality of life outcome is associated with cessation rather than reduction of psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 3(5), 455-459.

Quinn, M.C., Schofield, M., & Middleton, W. (2008). Conceptualization and treatment of psychogenic non-epileptic seizures. *Journal of Trauma & Dissociation*, 9(1), 63-84. doi:10.1080/15299730802073676

Quinn, M. C., Schofield, M. J., & Middleton, W. (2010). Permission to speak: Therapists' understandings of psychogenic nonepileptic seizures and their treatment. *Journal of Trauma & Dissociation*, 11(1), 108-123. doi:10.1080/15299730903491322

Quinn, M. C., Schofield, M. J., & Middleton, W. (2012). Successful psychotherapy for psychogenic seizures in men. *Psychotherapy Research*, 22(6), 682-698. doi:10.1080/10503307.2012.704085

Radford, L., Corral, S., Bradley, C., Fisher, H., Bassett, C., Howat, N., & Collishaw, S. (2011). *Child abuse and neglect in the UK today*. London: NSPCC.

Ramani, S. V., Quesney, L. F., Olson, D., & Gumnit, R. J. (1980). Diagnosis of hysterical seizures in epileptic patients. *The American Journal of Psychiatry*, 137(6), 705-709.

Ramos, J. A., & Brull, S. J. (2013). Psychogenic non-epileptic seizures in the post-anesthesia recovery unit. *Brazilian Journal of Anesthesiology*, (0) doi:http://dx.doi.org/10.1016/j.bjane.2013.10.005

Rao, M. L., Stefan, H., & Bauer, J. (1989). Epileptic but not psychogenic seizures are accompanied by simultaneous elevation of serum pituitary hormones and cortisol levels. *Neuroendocrinology*, 49(1), 33-39.

Rauch, S. A., Eftekhari, A., & Ruzek, J. I. (2012). Review of exposure therapy: A gold standard for PTSD treatment. *Journal of Rehabilitation Research and Development*, 49(5), 679-687.

Rawat, V. S., Dhiman, V., Sinha, S., Vijay Sagar, K. J., Thippeswamy, H., Chaturvedi, S. K., . . . Satishchandra, P. (2014). Co-morbidities and outcome of childhood psychogenic non-epileptic seizures--an observational study. *Seizure*, doi:S1059-1311(14)00262-3 [pii]

Razali, N. M., & Wah, Y. B. (2011). Power comparisons of shapiro-wilk, kolmogorov-smirnov, lilliefors and anderson-darling tests. *Journal of Statistical Modeling and Analytics*, 2(1), 21-33.

Razvi, S., Mulhern, S., & Duncan, R. (2012). Newly diagnosed psychogenic nonepileptic seizures: Health care demand prior to and following diagnosis at a first seizure clinic. *Epilepsy & Behavior*, 23(1), 7-9. doi:http://dx.doi.org/10.1016/j.yebeh.2011.10.009

Rechlin, T., Loew, T. H., & Joraschky, P. (1997). Pseudoseizure "status". *Journal of Psychosomatic Research*, 42(5), 495-498.

Reeves, A. L., McAuley, J. W., Moore, J. L., & Capestany, J. (1998). Medication use, self-reported drug allergies, and estimated medication cost in patients with epileptic versus nonepileptic seizures. *Journal of Epilepsy*, 11(4), 191-194.  
doi:http://dx.doi.org/10.1016/S0896-6974%2898%2900015-2

Reijs, R., Aldenkamp, A. P., & De Krom, M. (2004). Mood effects of antiepileptic drugs. *Epilepsy & Behavior*, 5, 66-76.

Reilly, J., Baker, G. A., Rhodes, J., & Salmon, P. (1999). The association of sexual and physical abuse with somatization: Characteristics of patients presenting with irritable bowel syndrome and non-epileptic attack disorder. *Psychological Medicine*, 29(2), 399-406.

Reinsberger, C., Perez, D. L., Murphy, M. M., & Dworetzky, B. A. (2012). Pre- and postictal, not ictal, heart rate distinguishes complex partial and psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 23(1), 68-70. doi:10.1016/j.yebeh.2011.10.008

Reuber, M. (2008). Psychogenic nonepileptic seizures: Answers and questions. *Epilepsy & Behavior*, 12(4), 622-635. doi:http://dx.doi.org/10.1016/j.yebeh.2007.11.006

Reuber, M. (2009). The etiology of psychogenic non-epileptic seizures: Toward a biopsychosocial model. *Neurologic Clinics*, 27(4), 909-+. doi:10.1016/j.ncl.2009.06.004

Reuber, M., Baker, G. A., Gill, R., Smith, D. F., & Chadwick, D. W. (2004). Failure to recognize psychogenic nonepileptic seizures may cause death. *Neurology*, 62(5), 834-835.

Reuber, M., Burness, C., Howlett, S., Brazier, J., & Grunewald, R. (2007). Tailored psychotherapy for patients with functional neurological symptoms: A pilot study. *Journal of Psychosomatic Research*, 63(6), 625-632. doi:10.1016/j.jpsychores.2007.06.013

Reuber, M., Fernández, G., Bauer, J., Helmstaedter, C., & Elger, C. E. (2002). Delays in the diagnosis of psychogenic non-epileptic seizures. *Journal of Neurology Neurosurgery and Psychiatry*, 73(2), 224-224.

Reuber, M., Fernández, G., Bauer, J., Singh, D. D., & Elger, C. E. (2002). Interictal EEG abnormalities in patients with psychogenic nonepileptic seizures. *Epilepsia*, 43(9), 1013-1020.

Reuber, M., Fernández, G., Helmstaedter, C., Qurishi, A., & Elger, C. E. (2002). Evidence of brain abnormality in patients with psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 3(3), 249-254. doi:10.1016/S1525-5050(02)00004-5

Reuber, M., & House, A. O. (2002). Treating patients with psychogenic non-epileptic seizures. *Current Opinion in Neurology*, 15(2), 207-211. doi:10.1097/00019052-200204000-00014

Reuber, M., House, A. O., Pukrop, R., Bauer, J., & Elger, C. E. (2003). Somatization, dissociation and general psychopathology in patients with psychogenic non-epileptic seizures. *Epilepsy Research*, 57(2-3), 159-167. doi:10.1016/j.eplepsyres.2003.11.004

Reuber, M., Howlett, S., & Kemp, S. (2005). Psychologic treatment of patients with psychogenic nonepileptic seizures. *Expert Review of Neurotherapeutics*, 5(6), 737-52. doi:10.1586/14737175.5.6.737



Reuber, M., Howlett, S., Khan, A., & Grunewald, R. A. (2007). Non-epileptic seizures and other functional neurological symptoms: Predisposing, precipitating, and perpetuating factors. *Psychosomatics*, 48(3), 230-238. doi:10.1176/appi.psy.48.3.230

Reuber, M., Jamnadas-Khoda, J., Broadhurst, M., Grunewald, R., Howell, S., Koepp, M., . . . Walker, M. (2011). Psychogenic nonepileptic seizure manifestations reported by patients and witnesses. *Epilepsia*, 52(11), 2028-2035. doi:10.1111/j.1528-1167.2011.03162.x

Reuber, M., Kral, T., Kurthen, M., & Elger, C. E. (2002). New-onset psychogenic seizures after intracranial neurosurgery. *Acta Neurochirurgica*, 144(9), 901-907. doi:http://dx.doi.org/10.1007/s00701-002-0993-7

Reuber, M., & Kurthen, M. (2011). Consciousness in non-epileptic attack disorder. *Behavioural Neurology*, 24(1), 95-106. doi:10.3233/BEN-2011-0315

Reuber, M., Kurthen, M., Fernández, G., Schramm, J., & Elger, C. E. (2002). Epilepsy surgery in patients with additional psychogenic seizures. *Archives of Neurology*, 59(1), 82-86.

Reuber, M., & Plug, L. (2009). Analysis of seizure metaphors differentiates between epileptic and nonepileptic seizures. *Epilepsia*, 50, 150. doi:http://dx.doi.org/10.1111/j.1528-1167.2009.02063.x

Reuber, M., Pukrop, R., Bauer, J., Derfuss, R., & Elger, C. E. (2004). Multidimensional assessment of personality in patients with psychogenic non-epileptic seizures. *Journal of Neurology, Neurosurgery, and Psychiatry*, 75(5), 743-748.

Reuber, M., Pukrop, R., Bauer, J., Helmstaedter, C., Tessendorf, N., & Erich Elger, C. (2003). Outcome in psychogenic nonepileptic seizures: 1 to 10-year follow-up in 164 patients. *Annals of Neurology*, 53(3), 305-311. doi:http://dx.doi.org/10.1002/ana.3000

Reuber, M., Pukrop, R., Mitchell, A. J., Bauer, J., & Elger, C. E. (2003). Clinical significance of recurrent psychogenic nonepileptic seizure status. *Journal of Neurology*, 250(11), 1355-1362.

Reuber, M., Monzoni, C., Sharrack, B., & Plug, L. (2009). Using conversation analysis to distinguish between epilepsy and non-epileptic seizures: A prospective multi-rater study. *Epilepsia*, 50, 43-43.

Riaz, H., Comish, S., Lawton, L., & Scheepers, B. (1998). Non-epileptic attack disorder and clinical outcome: A pilot study. *Seizure*, 7(5), 365-368. doi:10.1016/S1059-1311(05)80004-4

Ribai, P., Tugendhaft, P., & Legros, B. (2006). Usefulness of prolonged video-EEG monitoring and provocative procedure with saline injection for the diagnosis of non epileptic seizures of psychogenic origin. *Journal of Neurology*, 253(3), 328-332. doi:10.1007/s00415-005-0991-9

Roberts, N. A., Burleson, M. H., Weber, D. J., Larson, A., Sergeant, K., Devine, M. J., . . . Wang, N. C. (2012). Emotion in psychogenic nonepileptic seizures: Responses to affective pictures. *Epilepsy & Behavior*, 24(1), 107-115. doi:http://dx.doi.org/10.1016/j.yebeh.2012.03.018

Roberts, N. A., & Reuber, M. (2014). Alterations of consciousness in psychogenic nonepileptic seizures: Emotion, emotion regulation and dissociation. *Epilepsy and Behavior*, 30, 43-49. doi:http://dx.doi.org/10.1016/j.yebeh.2013.09.035

Robson, C., Drew, P., Walker, T., & Reuber, M. (2012). Catastrophising and normalising in patient's accounts of their seizure experiences. *Seizure*, 21(10), 795-801. doi:10.1016/j.seizure.2012.09.007

Rodewald, F., Dell, P. F., Wilhelm-Gößling, C., & Gast, U. (2010). Are major dissociative disorders characterized by a qualitatively different kind of dissociation? *Journal of Trauma & Dissociation*, 12(1), 9-24.

Rodin, G., de Groot, J., & Spivak, H. (2002). Trauma, dissociation, and somatization. In J. D. Bremner, & C. R. Marmar (Eds.), *Trauma, memory, and dissociation* (pp. 161-178). Washington: American Psychiatric Press.

Rodríguez-Urrutia, A., Toledo, M., Eiroa-Orosa, F. J., Salas-Puig, X., Santamarina, E., Fidel-Kinori, S. G., . . . Casas, M. (2014). Psychosocial factors and antiepileptic drug use related to delayed diagnosis of refractory psychogenic nonepileptic seizures. *Cognitive and Behavioral Neurology*, 27(4), 199-205.

Roelofs, K., Keijsers, G. P., Hoogduin, K. A., Naring, G. W., & Moene, F. C. (2002). Childhood abuse in patients with conversion disorder. *The American Journal of Psychiatry*, 159(11), 1908-1913.

Roelofs, J., Peters, M. L., Zeegers, M., & Vlaeyen, J. W. (2002). The modified Stroop paradigm as a measure of selective attention towards pain-related stimuli among chronic pain patients: A meta-analysis. *European Journal of Pain*, 6(4), 273-281.

Roelofs, K., & Spinhoven, P. (2007). Trauma and medically unexplained symptoms towards an integration of cognitive and neuro-biological accounts. *Clinical Psychology Review*, 27(7), 798-820. doi:S0272-7358(07)00125-0 [pii]

Rosemergy, I., Frith, R., Herath, S., & Walker, E. (2013). Use of postictal respiratory pattern to discriminate between convulsive psychogenic nonepileptic seizures and generalized tonic-clonic seizures. *Epilepsy and Behavior*, 27(1), 81-84.  
doi:http://dx.doi.org/10.1016/j.yebeh.2012.12.024

Rosenbaum, M. (2000). Psychogenic seizures - why women? *Psychosomatics*, 41(2), 147-149. doi:10.1176/appi.psy.41.2.147

Rosenberg, H. J., Rosenberg, S. D., Williamson, P. D., & Wolford, G. L. (2000). A comparative study of trauma and posttraumatic stress disorder prevalence in epilepsy patients and psychogenic nonepileptic seizure patients. *Epilepsia*, 41(4), 447-452.  
doi:10.1111/j.1528-1157.2000.tb00187.x

Ross, C. A., Miller, S. D., Bjornson, L., & Reagor, P. (1991). Abuse histories in 102 cases of multiple personality disorder. *The Canadian Journal of Psychiatry/La Revue Canadienne De Psychiatrie*, 36(2), 97-101.

Ross, C. A., & Ness, L. (2010). Symptom patterns in dissociative identity disorder patients and the general population. *Journal of Trauma & Dissociation*, 11(4), 458-468.

Rotge, J., Lambrecq, V., Marchal, C., Pedespan, J., Burbaud, P., Rougier, A., & Michel, V. (2009). Conversion disorder and coexisting nonepileptic seizures in patients with refractory seizures. *Epilepsy & Behavior*, 16(2), 350-352.  
doi:<http://dx.doi.org/10.1016/j.yebeh.2009.07.032>

Rotter, J. B. (1990). Internal versus external control of reinforcement: A case history of a variable. *American Psychologist*, 45(4), 489.

Sack, M., Boroske-Leiner, K., & Lahmann, C. (2010). Association of nonsexual and sexual traumatizations with body image and psychosomatic symptoms in psychosomatic outpatients. *General Hospital Psychiatry*, 32(3), 315-320.  
doi:10.1016/j.genhosppsych.2010.01.002 [doi]

Sahaya, K., Dholakia, S., Lardizabal, D., & Sahota, P. K. (2011). Opinion survey of health care providers towards psychogenic non epileptic seizures. *Clinical neurology & neurosurgery*, 114(10), 1304-1307.

Salinsky, M., Evrard, C., Storzbach, D., & Pugh, M. J. (2012). Psychiatric comorbidity in veterans with psychogenic seizures. *Epilepsy & Behavior*, 25(3), 345-349.  
doi:<http://dx.doi.org/10.1016/j.yebeh.2012.07.013>

Salinsky, M., Spencer, D., Boudreau, E., & Ferguson, F. (2011). Psychogenic nonepileptic seizures in US veterans. *Neurology*, 77(10), 945-950.  
doi:10.1212/WNL.0b013e31822cfc46

- Salmon, P., Al-Marzooqi, S. M., Baker, G., & Reilly, J. (2003). Childhood family dysfunction and associated abuse in patients with nonepileptic seizures: Towards a causal model. *Psychosomatic Medicine*, 65(4), 695-700.  
doi:10.1097/01.PSY.0000075976.20244.D8
- Salpekar, J. A., Plioplys, S., Siddarth, P., Bursch, B., Shaw, R. J., Asato, M. R., . . . Caplan, R. (2010). Pediatric psychogenic nonepileptic seizures: A study of assessment tools. *Epilepsy & Behavior*, 17(1), 50-55. doi:http://dx.doi.org/10.1016/j.yebeh.2009.10.002
- Sar, V., Koyuncu, A., Ozturk, E., Yargic, L. I., Kundakci, T., Yazici, A., . . . Aksut, D. (2007). Dissociative disorders in the psychiatric emergency ward. *General Hospital Psychiatry*, 29(1), 45-50. doi:S0163-8343(06)00211-8 [pii]
- Sar, V., Tutkun, H., Alyanak, B., Bakim, B., & Baral, I. (2000). Frequency of dissociative disorders among psychiatric outpatients in Turkey. *Comprehensive Psychiatry*, 41(3), 216-222. doi:S0010-440X(00)90050-6 [pii]
- Sauer, C., Arens, E. A., Stopsack, M., Spitzer, C., & Barnow, S. (2014). Emotional hyper-reactivity in borderline personality disorder is related to trauma and interpersonal themes. *Psychiatry Research*, 220(1-2), 468-476. doi:10.1016/j.psychres.2014.06.041 [doi]
- Say, G. N., Tasdemir, H. A., Akbas, S., Yuce, M., & Karabekiroglu, K. (2014). Self-esteem and psychiatric features of Turkish adolescents with psychogenic non-epileptic seizures: A comparative study with epilepsy and healthy control groups. *International Journal of Psychiatry in Medicine*, 47(1), 41-53. doi:http://dx.doi.org/10.2190/PM.47.1.d
- Saygi, S., Katz, A., Marks, D. A., & Spencer, S. S. (1992). Frontal lobe partial seizures and psychogenic seizures: Comparison of clinical and ictal characteristics. *Neurology*, 42(7), 1274-1277.
- Scerbo, A. S., Freedman, L. W., Raine, A., Dawson, M. E., & Venables, P. H. (1992). A major effect of recording site on measurement of electrodermal activity. *Psychophysiology*, 29(2), 241-246.

- Scévola, L., Teitelbaum, J., Oddo, S., Centurion, E., Loidl, C. F., Kochen, S., & DDAlessio, L. (2013). Psychiatric disorders in patients with psychogenic nonepileptic seizures and drug-resistant epilepsy: A study of an argentine population. *Epilepsy & Behavior*, 29(1), 155-160. doi:http://dx.doi.org/10.1016/j.yebeh.2013.07.012
- Schachter, S. C., Brown, F., & Rowan, A. J. (1996). Provocative testing for nonepileptic seizures: Attitudes and practices in the United States among American Epilepsy Society members. *Journal of Epilepsy*, 9(4), 249-252.
- Scheepers, B., Budd, S., Curry, S., Gregory, S., & Elson, S. (1994). Non-epileptic attack disorder: A clinical audit. *Seizure*, 3(2), 129-34. doi:10.1016/S1059-1311(05)80203-1
- Schmahl, C. G., Vermetten, E., Elzinga, B. M., & Bremner, J. D. (2004). A positron emission tomography study of memories of childhood abuse in borderline personality disorder. *Biological Psychiatry*, 55(7), 759-765.
- Schmider, E., Ziegler, M., Danay, E., Beyer, L., & Bühner, M. (2010). Is it really robust? Reinvestigating the robustness of ANOVA against violations of the normal distribution assumption. *European Journal of Research Methods for the Behavioral and Social Sciences*, 6(4), 147-151.
- Schmitz, B. (2010). Psychogenic nonepileptic seizures: Why women? In Schachter, S.C., & LaFrance Jr, W.C. (Ed.), *Gates and Rowan's nonepileptic seizures* (3rd ed., pp. 131-135). Cambridge: Cambridge University Press.
- Schmutz, M. (2013). Dissociative seizures - A critical review and perspective. *Epilepsy & Behavior*, 29(3), 449-456. doi:10.1016/j.yebeh.2013.08.018; 10.1016/j.yebeh.2013.08.018
- Schramke, C. J., Kay, K. A., Valerian, J. P., & Kelly, K. M. (2010). Using patient history to distinguish between patients with non-epileptic and patients with epileptic events. *Epilepsy & Behavior*, 19(3), 478-482. doi:10.1016/j.yebeh.2010.08.003

Schramke, C. J., Valeri, A., Valeriano, J. P., & Kelly, K. M. (2007). Using the Minnesota Multiphasic Inventory 2, EEGs, and clinical data to predict nonepileptic events. *Epilepsy & Behavior*, 11(3), 343-346. doi:<http://dx.doi.org/10.1016/j.yebeh.2007.06.011>

Schwabe, M., Howell, S. J., & Reuber, M. (2007). Differential diagnosis of seizure disorders: A conversation analytic approach. *Social Science & Medicine*, 65(4), 712-724. doi:<http://dx.doi.org/10.1016/j.socscimed.2007.03.045>

Schwarz, B. E., Bickford, R. G., & Rasmussen, W. C. (1955). Hypnotic phenomena, including hypnotically activated seizures, studied with the electroencephalogram. *The Journal of Nervous and Mental Disease*, 122(6), 564-574.

Scull, D. A. (1997). Pseudoseizures or non-epileptic seizures (NES); 15 synonyms. *Journal of Neurology, Neurosurgery, and Psychiatry*, 62(2), 200.

Selbst, S. M., & Clancy, R. (1996). Pseudoseizures in the pediatric emergency department. *Pediatric Emergency Care*, 12(3), 185-188.

Selkirk, M., Duncan, R., Oto, M., & Pelosi, A. (2008). Clinical differences between patients with nonepileptic seizures who report antecedent sexual abuse and those who do not. *Epilepsia*, 49(8), 1446-1450. doi:<http://dx.doi.org/10.1111/j.1528-1167.2008.01611.x>

Selwa, L. M., Geyer, J., Nikakhtar, N., Brown, M. B., Schuh, L. A., & Drury, I. (2000). Nonepileptic seizure outcome varies by type of spell and duration of illness. *Epilepsia*, 41(10), 1330-1334.

Sen, A., Scott, C., & Sisodiya, S. M. (2007). Stertorous breathing is a reliably identified sign that helps in the differentiation of epileptic from psychogenic non-epileptic convulsions: An audit. *Epilepsy Research*, 77(1), 62-64. doi:10.1016/j.eplepsyres.2007.07.009

- Seneviratne, U., Briggs, B., Lowenstern, D., & D'Souza, W. (2011). The spectrum of psychogenic non-epileptic seizures and comorbidities seen in an epilepsy monitoring unit. *Journal of Clinical Neuroscience*, 18(3), 361-363.
- Seneviratne, U., Rahman, Z., Diamond, A., & Brusco, M. (2012). The yield and clinical utility of outpatient short-term video-electroencephalographic monitoring: A five-year retrospective study. *Epilepsy & Behavior*, 25(3), 303-306.  
doi:10.1016/j.yebeh.2012.08.018; 10.1016/j.yebeh.2012.08.018
- Seneviratne, U., Reutens, D., & D'Souza, W. (2010). Stereotypy of psychogenic nonepileptic seizures: Insights from video-EEG monitoring. *Epilepsia*, 51(7), 1159-1168.  
doi:10.1111/j.1528-1167.2010.02560.x
- Sethi, N. K. (2013). Psychogenic nonepileptic seizures: The name matters. *JAMA Neurology*, 70(4), 528-529. doi:http://dx.doi.org/10.1001/jamaneurol.2013.1898
- Shaffer, J. P. (1995). Multiple hypothesis testing. *Annual Review of Psychology*, 46(1), 561-584.
- Sharpe, D., & Faye, C. (2006). Non-epileptic seizures and child sexual abuse: A critical review of the literature. *Clinical Psychology Review*, 26(8), 1020-1040.  
doi:10.1016/j.cpr.2005.11.011
- Shen, W., Bowman, E. S., & Markand, O. N. (1990). Presenting the diagnosis of pseudoseizure. *Neurology*, 40(5), 756-759.
- Shin, L. M., Wright, C. I., Cannistraro, P. A., Wedig, M. M., McMullin, K., Martis, B., . . . Krangel, T. S. (2005). A functional magnetic resonance imaging study of amygdala and medial prefrontal cortex responses to overtly presented fearful faces in posttraumatic stress disorder. *Archives of General Psychiatry*, 62(3), 273-281.
- Shneker, B. F., & Elliott, J. O. (2008). Primary care and emergency physician attitudes and beliefs related to patients with psychogenic nonepileptic spells. *Epilepsy & Behavior*, 13(1), 243-247. doi:10.1016/j.yebeh.2008.03.001



Sierra, M., Senior, C., Dalton, J., McDonough, M., Bond, A., Phillips, M. L., . . . David, A. S. (2002). Autonomic response in depersonalization disorder. *Archives of General Psychiatry*, 59(9), 833-838. .

Sierra, M., Senior, C., Phillips, M. L., & David, A. S. (2006). Autonomic response in the perception of disgust and happiness in depersonalization disorder. *Psychiatry Research*, 145(2), 225-231.

Sieswerda, S., Arntz, A., Mertens, I., & Vertommen, S. (2007). Hypervigilance in patients with borderline personality disorder: Specificity, automaticity, and predictors. *Behaviour Research and Therapy*, 45(5), 1011-1024.

Sigurdardottir, K. R., & Olafsson, E. (1998). Incidence of psychogenic seizures in adults: A population-based study in Iceland. *Epilepsia*, 39(7), 749-752.

Silva, W., Giagante, B., Saizar, R., D'Alessio, L., Oddo, S., Consalvo, D., . . . Kochen, S. (2001). Clinical features and prognosis of nonepileptic seizures in a developing country. *Epilepsia*, 42(3), 398-401. doi:<http://dx.doi.org/10.1046/j.1528-1157.2001.45299.x>

Silvers, J. A., McRae, K., Gabrieli, J. D., Gross, J. J., Remy, K. A., & Ochsner, K. N. (2012). Age-related differences in emotional reactivity, regulation, and rejection sensitivity in adolescence. *Emotion*, 12(6), 1235.

Sirven, J. I., & Glosser, D. S. (1998). Psychogenic nonepileptic seizures: Theoretic and clinical considerations. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, 11(4), 225-235.

Slater, J. D., Brown, M. C., Jacobs, W., & Ramsay, R. E. (1995). Induction of pseudoseizures with intravenous saline placebo. *Epilepsia*, 36(6), 580-585.

Slavney, P. R. (1994). In defense of pseudoseizure. *General Hospital Psychiatry*, 16(4), 243-245. doi:[http://dx.doi.org/10.1016/0163-8343\(94\)90001-9](http://dx.doi.org/10.1016/0163-8343(94)90001-9)

Smith, J. C., Bradley, M. M., & Lang, P. J. (2005). State anxiety and affective physiology: Effects of sustained exposure to affective pictures. *Biological Psychology*, 69(3), 247-260.

Smith, J. A., & Osborn, M. (2008). Interpretative Phenomenological Analysis. In J. A. Smith (Ed.), *Qualitative psychology: A practical guide to research methods* (2nd ed., pp. 53-80). London: Sage.

Smith, M. L., Stagno, S. J., Dolske, M., Kosalko, J., McConnell, C., Kaspar, L., & Lederman, R. (1997). Induction procedures for psychogenic seizures: Ethical and clinical considerations. *The Journal of Clinical Ethics*, 8(3), 217-229.

Snyder, S. L., Rosenbaum, D. H., Rowan, A. J., & Strain, J. J. (1994). SCID diagnosis of panic disorder in psychogenic seizure patients. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 6(3), 261-266.

Specht, U., & Thorbecke, R. (2009). Should patients with psychogenic nonepileptic seizures be allowed to drive? Recommendations of German experts. *Epilepsy & Behavior*, 16(3), 547-550. doi:10.1016/j.yebeh.2009.09.012; 10.1016/j.yebeh.2009.09.012

Spence, S. A. (2006). All in the mind? The neural correlates of unexplained physical symptoms. *Advances in Psychiatric Treatment*, 12(5), 349-358.

Spinhoven, P., Roelofs, K., Moene, F., Kuyk, J., Nijenhuis, E., Hoogduin, K., & Van Dyck, R. (2004). Trauma and dissociation in conversion disorder and chronic pelvic pain. *International Journal of Psychiatry in Medicine*, 34(4), 305-318. doi:10.2190/YDK2-C66W-CL6L-N5TK

Spitzer, C., Barnow, S., Freyberger, H. J., & Grabe, H. J. (2006). Recent developments in the theory of dissociation. *World Psychiatry*, 5(2), 82-86.

Stagno, S. J., & Smith, M. L. (1996). Use of induction procedures in diagnosing psychogenic seizures. *Journal of Epilepsy*, 9(3), 153-158. doi:10.1016/0896-6974(96)00002-3

Stanhope, N., Goldstein, L. H., & Kuipers, E. (2003). Expressed emotion in the relatives of people with epileptic or nonepileptic seizures. *Epilepsia*, 44(8), 1094-1102.  
doi:10.1046/j.1528-1157.2003.09503.x

Staniloiu, A., Markowitsch, H. J., & Vitcu, I. (2012). Neuroimaging and dissociative disorders. In V. Chaudhury (Ed.), *Advances in brain imaging* (pp.11-34). Rijeka, Croatia: INTECH Open Access Publisher.

Staudenmayer, H., & Kramer, R. E. (1999). Psychogenic chemical sensitivity: Psychogenic pseudoseizures elicited by provocation challenges with fragrances. *Journal of Psychosomatic Research*, 47(2), 185-190.

Steinberg, M., Cicchetti, D., Buchanan, J., & Hall, P. (1993). Clinical assessment of dissociative symptoms and disorders: The Structured Clinical Interview for DSM-IV Dissociative Disorders (SCID-D). *Dissociation: Progress in the Dissociative Disorders*, 6 (1), 3-15.

Stevens, J. S., & Hamann, S. (2012). Sex differences in brain activation to emotional stimuli: A meta-analysis of neuroimaging studies. *Neuropsychologia*, 50(7), 1578-1593.  
doi:10.1016/j.neuropsychologia.2012.03.011 [doi]

Stewart, R. S., Lovitt, R., & Stewart, R. M. (1982). Are hysterical seizures more than hysteria? A research diagnostic criteria, DMS-III, and psychometric analysis. *The American Journal of Psychiatry*, 139(7), 926-929.

Stone, J. (2006). Dissociation: What is it and why is it important? *Practical Neurology*, 6(5), 308-313.

Stone, J., Binzer, M., & Sharpe, M. (2004). Illness beliefs and locus of control: A comparison of patients with pseudoseizures and epilepsy. *Journal of Psychosomatic Research*, 57(6), 541-547.

Stone, J., Campbell, K., Sharma, N., Carson, A., Warlow, C. P., & Sharpe, M. (2003). What should we call pseudoseizures? The patient's perspective. *Seizure*, 12(8), 568-572.

Stone, J., & Carson, A. J. (2013). The unbearable lightheadedness of seizing: Wilful submission to dissociative (non-epileptic) seizures. *Journal of Neurology, Neurosurgery and Psychiatry*, 84(7), 822-824. doi:<http://dx.doi.org/10.1136/jnnp-2012-304842>

Stone, J., Sharpe, M., & Binzer, M. (2004). Motor conversion symptoms and pseudoseizures: A comparison of clinical characteristics. *Psychosomatics*, 45(6), 492-499. doi:10.1176/appi.psy.45.6.492

Stone, J., Zeman, A., Simonotto, E., Meyer, M., Azuma, R., Flett, S., & Sharpe, M. (2007). FMRI in patients with motor conversion symptoms and controls with simulated weakness. *Psychosomatic Medicine*, 69(9), 961-969. doi:PSY.0b013e31815b6c14 [pii]

Storzbach, D., Binder, L. M., Salinsky, M. C., Campbell, B. R., & Mueller, R. M. (2000). Improved prediction of nonepileptic seizures with combined MMPI and EEG measures. *Epilepsia*, 41(3), 332-337. doi:10.1111/j.1528-1157.2000.tb00164.x

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology*, 18(6), 643.

Strutt, A. M., Hill, S. W., Scott, B. M., Uber-Zak, L., & Fogel, T. G. (2011a). A comprehensive neuropsychological profile of women with psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 20(1), 24-28. doi:10.1016/j.yebeh.2010.10.004

Strutt, A. M., Hill, S. W., Scott, B. M., Uber-Zak, L., & Fogel, T. G. (2011b). Motivation, psychopathology, locus of control, and quality of life in women with epileptic and nonepileptic seizures. *Epilepsy & Behavior*, 22(2), 279-284. doi:<http://dx.doi.org/10.1016/j.yebeh.2011.06.020>

Sullivan, S., Ruffman, T., & Hutton, S. B. (2007). Age differences in emotion recognition skills and the visual scanning of emotion faces. *The Journals of Gerontology. Series B, Psychological Sciences and Social Sciences*, 62(1), P53-60. doi:62/1/P53 [pii]

Swingle, P. G. (1998). Neurofeedback treatment of pseudoseizure disorder. *Biological Psychiatry*, 44(11), 1196-1199.

Syed, T. U., Arozullah, A. M., Loparo, K. L., Jamasebi, R., Suci, G. P., Griffin, C., . . . Alexopoulos, A. V. (2009). A self-administered screening instrument for psychogenic nonepileptic seizures. *Neurology*, 72(19), 1646-1652.  
doi:10.1212/WNL.0b013e3181a55ef7; 10.1212/WNL.0b013e3181a55ef7

Syed, T. U., LaFrance Jr, W.C, Kahriman, E. S., Hasan, S. N., Rajasekaran, V., . . . Koubeissi, M. Z. (2011). Can semiology predict psychogenic nonepileptic seizures? a prospective study. *Annals of Neurology*, 69(6), 997-1004.  
doi:http://dx.doi.org/10.1002/ana.22345

Szabo, L., Siegler, Z., Zubek, L., Liptai, Z., Korhegyi, I., Bansagi, B., & Fogarasi, A. (2012). A detailed semiologic analysis of childhood psychogenic nonepileptic seizures. *Epilepsia*, 53(3), 565-570. doi:http://dx.doi.org/10.1111/j.1528-1167.2012.03404.x

Szaflarski, J. P., Ficker, D. M., Cahill, W. T., & Privitera, M. D. (2000). Four-year incidence of psychogenic nonepileptic seizures in adults in Hamilton county, OH. *Neurology*, 55(10), 1561-1563.

Szaflarski, J. P., Hughes, C., Szaflarski, M., Ficker, D. M., Cahill, W. T., Li, M., & Privitera, M. D. (2003). Quality of life in psychogenic nonepileptic seizures. *Epilepsia*, 44(2), 236-242.

Szaflarski, J. P., & Szaflarski, M. (2004). Seizure disorders, depression, and health-related quality of life. *Epilepsy & Behavior*, 5(1), 50-57.  
doi:http://dx.doi.org/10.1016/j.yebeh.2003.10.015

Tabachnick, B. G., & Fidell, L. S. (2007). *Using multivariate statistics* (5th ed.). Boston: Pearson Higher Education.

Tamietto, M., & De Gelder, B. (2010). Neural bases of the non-conscious perception of emotional signals. *Nature Reviews Neuroscience*, 11(10), 697-709.

- Tasca, C., Rapetti, M., Carta, M. G., & Fadda, B. (2012). Women and hysteria in the history of mental health. *Clinical Practice and Epidemiology in Mental Health*, 8, 110-119. doi:10.2174/1745017901208010110 [doi]
- Testa, S. M., & Brandt, J. (2010). Do patients with psychogenic nonepileptic seizures have positive covert attitudes toward sickness? *Epilepsy & Behavior*, 19(3), 323-327. doi:10.1016/j.yebeh.2010.07.014
- Testa, S. M., Krauss, G. L., Lesser, R. P., & Brandt, J. (2012). Stressful life event appraisal and coping in patients with psychogenic seizures and those with epilepsy. *Seizure*, 21(4), 282-287. doi:http://dx.doi.org/10.1016/j.seizure.2012.02.002
- Testa, S. M., Lesser, R. P., Krauss, G. L., & Brandt, J. (2011). Personality Assessment Inventory among patients with psychogenic seizures and those with epilepsy. *Epilepsia*, 52(8), E84-E88. doi:10.1111/j.1528-1167.2011.03141.x
- Testa, S. M., Schefft, B. K., Szaflarski, J. P., Yeh, H. S., & Privitera, M. D. (2007). Mood, personality, and health-related quality of life in epileptic and psychogenic seizure disorders. *Epilepsia*, 48(5), 973-982. doi:10.1111/j.1528-1167.2006.00965.x
- Thomas, A. A., Preston, J., Scott, R. C., & Bujarski, K. A. (2013). Diagnosis of probable psychogenic nonepileptic seizures in the outpatient clinic: Does gender matter? *Epilepsy and Behavior*, 29(2), 295-297. doi:http://dx.doi.org/10.1016/j.yebeh.2013.08.006
- Thompson, A. W., Hantke, N., Phatak, V., & Chaytor, N. (2010). The Personality Assessment Inventory as a tool for diagnosing psychogenic nonepileptic seizures. *Epilepsia*, 51(1), 161-164. doi:http://dx.doi.org/10.1111/j.1528-1167.2009.02151.x
- Thompson, N. C., Osorio, I., & Hunter, E. E. (2005). Nonepileptic seizures: Reframing the diagnosis. *Perspectives in Psychiatric Care*, 41(2), 71-78. doi:10.1111/j.1744-6163.2005.00023.x

Thompson, N.C., Connelly, L., Peltzer, J., Nowack, W. J., Hamera, E., & Hunter, E. E. (2013). Psychogenic nonepileptic seizures: A pilot study of a brief educational intervention. *Perspectives in Psychiatric Care*, 49(2), 78-83. doi:10.1111/j.1744-6163.2012.00353.x

Thompson, P. M., Batzel, L. W., & Wilkus, R. J. (1992). Millon Clinical Multiaxial Inventory assessments of patients manifesting either psychogenic or epileptic seizures. *Journal of Epilepsy*, 5(4), 226-230. doi:10.1016/S0896-6974(05)80121-5

Thompson, R., Isaac, C. L., Rowse, G., Tooth, C. L., & Reuber, M. (2009). What is it like to receive a diagnosis of nonepileptic seizures? *Epilepsy & Behavior*, 14(3), 508-515.

Tojek, T., Lumley, M., Barkley, G., Mahr, G., & Thomas, A. (2000). Stress and psychosocial correlates of psychogenic non-epileptic seizures. *Psychosomatic Medicine*, 62(1), 122-123.

Trimble, M. (2010). Psychogenic nonepileptic seizures: Historical overview. In S. C. Schachter, & W. C. LaFrance Jr (Eds.), *Gates and Rowan's nonepileptic seizures* (3rd ed., pp. 17-26). Cambridge: Cambridge University Press.

Turner, K., Piazzini, A., Chiesa, V., Barbieri, V., Vignoli, A., Gardella, E., . . . Gambini, O. (2011). Patients with epilepsy and patients with psychogenic non-epileptic seizures: Video-EEG, clinical and neuropsychological evaluation. *Seizure*, 20(9), 706-710. doi:http://dx.doi.org/10.1016/j.seizure.2011.07.001

Twamley, E., & Bortz, J. (1999). Nonepileptic seizures: Neuropsychological mechanisms. *BNI Quarterly*, 15(1), 20-26.

Uliaszek, A. A., Prensky, E., & Baslet, G. (2012). Emotion regulation profiles in psychogenic non-epileptic seizures. *Epilepsy & Behavior*, 23(3), 364-369. doi:10.1016/j.yebeh.2012.01.009

Unoka, Z., Fogd, D., Füzy, M., & Csukly, G. (2011). Misreading the facial signs: Specific impairments and error patterns in recognition of facial emotions with negative valence in borderline personality disorder. *Psychiatry Research*, 189(3), 419-425.

Updyke, M., & Duryea, B. (2013). To provoke or not provoke: Ethical considerations in the epilepsy monitoring unit. *Journal of Neuroscience Nursing*, 45(3), 133-138.  
doi:10.1097/JNN.0b013e31828a414b

Urbanek, M., Harvey, M., McGowan, J., & Agrawal, N. (2014). Regulation of emotions in psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 37(0), 110-115.  
doi:http://dx.doi.org/10.1016/j.yebeh.2014.06.004

Van der Hart, O., & Horst, R. (1989). The dissociation theory of Pierre Janet. *Journal of Traumatic Stress*, 2(4), 397-412.

Van der Hart, O., Nijenhuis, E., Steele, K., & Brown, D. (2004). Trauma-related dissociation: Conceptual clarity lost and found. *The Australian and New Zealand Journal of Psychiatry*, 38(11-12), 906-914. doi:ANPI480 [pii]

Van der Kruijs, S. J. M., Bodde, N. M. G., & Aldenkamp, A. P. (2011). Psychophysiological biomarkers of dissociation in psychogenic non-epileptic seizures. *Acta Neurologica Belgica*, 111(2), 99-103.

Van der Kruijs, S. J. M., Bodde, N. M. G., Vaessen, M. J., Lazeron, R. H. C., Vonck, K., Boon, P., . . . Jansen, J. F. A. (2012). Functional connectivity of dissociation in patients with psychogenic non-epileptic seizures. *Journal of Neurology Neurosurgery and Psychiatry*, 83(3), 239-247. doi:10.1136/jnnp-2011-300776

Van der Kruijs, S. J. M., Jagannathan, S. R., Bodde, N. M. G., Besseling, R. M. H., Lazeron, R. H. C., Vonck, K. E. J., . . . Jansen, J. F. A. (2014). Resting-state networks and dissociation in psychogenic non-epileptic seizures. *Journal of Psychiatric Research*, 54(0), 126-133. doi:http://dx.doi.org/10.1016/j.jpsychires.2014.03.010



Van Merode, T., De Krom, M. C. T. F. M., & Knottnerus, J. A. (1997). Gender-related differences in non-epileptic attacks: A study of patients' cases in the literature. *Seizure*, 6(4), 311-316. doi:[http://dx.doi.org/10.1016/S1059-1311\(97\)80079-9](http://dx.doi.org/10.1016/S1059-1311(97)80079-9)

Van Merode, T., Twellaar, M., Kotsopoulos, I. A. W., Kessels, A. G. H., Merckelbach, H., de Krom, M. C. T. F. M., & Knottnerus, J. A. (2004). Psychological characteristics of patients with newly developed psychogenic seizures. *Journal of Neurology Neurosurgery and Psychiatry*, 75(8), 1175-1177. doi:10.1136/jnnp.2003.016923

Vancini, R. L., Arida, R. M., & de Lira, C. A. B. Can physical exercise be a coping strategy for psychological stress for patients with psychogenic seizures? *International Journal of Epilepsy*, (0) doi:<http://dx.doi.org/10.1016/j.ijep.2014.07.003>

Vanderlinden, J., Van Dyck, R., Vandereycken, W., Vertommen, H., & Jan Verkes, R. (1993). The Dissociation Questionnaire (DIS-Q): Development and characteristics of a new self-report questionnaire. *Clinical Psychology & Psychotherapy*, 1(1), 21-27.

Veague, H. B., & Hooley, J. M. (2014). Enhanced sensitivity and response bias for male anger in women with borderline personality disorder. *Psychiatry Research*, 215(3), 687-693.

Vincentiis, S., Valente, K. D., Thomé-Souza, S., Kuczinsky, E., Fiore, L. A., & Negrão, N. (2006). Risk factors for psychogenic nonepileptic seizures in children and adolescents with epilepsy. *Epilepsy & Behavior*, 8(1), 294-298.

Vinten, J., Adab, N., Kini, U., Gorry, J., Gregg, J., Baker, G. A., & Liverpool and Manchester Neurodevelopment Study Group. (2005). Neuropsychological effects of exposure to anticonvulsant medication in utero. *Neurology*, 64(6), 949-954. doi:10.1213/00006123-2005060000000949 [pii]

- Vinton, A., Carino, J., Vogrin, S., Macgregor, L., Kilpatrick, C., Matkovic, Z., & O'Brien, T. J. (2004). "Convulsive" nonepileptic seizures have a characteristic pattern of rhythmic artifact distinguishing them from convulsive epileptic seizures. *Epilepsia*, 45(11), 1344-1350. doi:10.1111/j.0013-9580.2004.04704.x
- Vittinghoff, E., & McCulloch, C. E. (2007). Relaxing the rule of ten events per variable in logistic and cox regression. *American Journal of Epidemiology*, 165(6), 710-718. doi:kwk052 [pii]
- Volow, M. R. (1986). Pseudoseizures - an overview. *Southern Medical Journal*, 79(5), 600-607. doi:10.1097/00007611-198605000-00020
- Vossler, D. G. (1995). Nonepileptic seizures of physiologic origin. *Journal of Epilepsy*, 8(1), 1-10. doi:http://dx.doi.org/10.1016/0896-6974%2894%2900014-Q
- Vossler, D. G., Haltiner, A. M., Schepp, S. K., Friel, P. A., Caylor, L. M., Morgan, J. D., & Doherty, M. J. (2004). Ictal stuttering: A sign suggestive of psychogenic nonepileptic seizures. *Neurology*, 63(3), 516-519.
- Vuilleumier, P. (2005). How brains beware: Neural mechanisms of emotional attention. *Trends in Cognitive Sciences*, 9(12), 585-594.
- Vuilleumier, P. (2014). Brain circuits implicated in psychogenic paralysis in conversion disorders and hypnosis. *Neurophysiologie Clinique/Clinical Neurophysiology*, 44(4), 323-337.
- Vuilleumier, P., & Schwartz, S. (2001). Emotional facial expressions capture attention. *Neurology*, 56(2), 153-158.
- Wadwekar, V., Nair, P. P., Murgai, A., Thirunavukkarasu, S., & Thazhath, H. K. (2014). Semiologic classification of psychogenic non epileptic seizures (PNES) based on video EEG analysis: Do we need new classification systems? *Seizure*, 23(3), 222-226. doi:http://dx.doi.org/10.1016/j.seizure.2013.12.005

Wagner, A. W., & Linehan, M. M. (1999). Facial expression recognition ability among women with borderline personality disorder: Implications for emotion regulation? *Journal of Personality Disorders*, 13(4), 329-344.

Wagner, M. T., Wymer, J. H., Topping, K. B., & Pritchard, P. B. (2005). Use of the Personality Assessment Inventory as an efficacious and cost-effective diagnostic tool for nonepileptic seizures. *Epilepsy and Behavior*, 7(2), 301-304.  
doi:<http://dx.doi.org/10.1016/j.yebeh.2005.05.017>

Walczak, T. S., Papacostas, S., Williams, D. T., Scheuer, M. L., Lebowitz, N., & Notarfrancesco, A. (1995). Outcome after diagnosis of psychogenic nonepileptic seizures. *Epilepsia*, 36(11), 1131-1137. doi:<http://dx.doi.org/10.1111/j.1528-1157.1995.tb00472.x>

Waller, G., Hamilton, K., Elliott, P., Lewendon, J., Stopa, L., Waters, A., . . . Kennerley, H. (2001). Somatoform dissociation, psychological dissociation, and specific forms of trauma. *Journal of Trauma & Dissociation*, 1(4), 81-98.

Waller, N., Putnam, F. W., & Carlson, E. B. (1996). Types of dissociation and dissociative types: A taxometric analysis of dissociative experiences. *Psychological Methods*, 1(3), 300.

Warrington, E. K., & James, M. (1991). *The Visual Object and Space Perception (VOSP) battery*. Bury St Edmunds: Thames Valley Test Company.

Wassmer, E., Wassmer, S. R., & Donati, F. (2003). Saline infusion: A diagnostic and therapeutic tool in nonepileptic attacks? *Epilepsy & Behavior*, 4(5), 500-506.  
doi:10.1016/S1525-5050(03)00181-1

Wechsler, D. (1997). *Wechsler Memory Scale - third edition (WMS-III)*. San Antonio, TX: Psychological Corporation.

Wechsler, D. (1999). *Wechsler Abbreviated Scale of Intelligence (WASI)*. San Antonio, TX: Psychological Corporation.

Wechsler, D. (2008). *Wechsler Adult Intelligence Scale—fourth edition (WAIS–IV)*. San Antonio, TX: Psychological Corporation.

Westbrook, L. E., Devinsky, O., & Geocadin, R. (1998). Nonepileptic seizures after head injury. *Epilepsia*, 39(9), 978-982. doi:<http://dx.doi.org/10.1111/j.1528-1157.1998.tb01447.x>

Whitaker, J. N., & Rosenberg, R. N. (2001). The confluence of quality of care, cost-effectiveness, pragmatism, and medical ethics in the diagnosis of nonepileptic seizures: A provocative situation for neurology. *Archives of Neurology*, 58(12), 2066-2067.

Whitehead, K., Kandler, R., & Reuber, M. (2013). Patients' and neurologists' perception of epilepsy and psychogenic nonepileptic seizures. *Epilepsia*, 54(4), 708-717. doi:<http://dx.doi.org/10.1111/epi.12087>

Whitehead, K., & Reuber, M. (2012). Illness perceptions of neurologists and psychiatrists in relation to epilepsy and non-epileptic attack disorder (NEAD). *Journal of Neurology Neurosurgery and Psychiatry*, 83(10) doi:10.1136/jnnp-2012-303538.38

Whittle, S., Yucel, M., Yap, M. B., & Allen, N. B. (2011). Sex differences in the neural correlates of emotion: Evidence from neuroimaging. *Biological Psychology*, 87(3), 319-333. doi:10.1016/j.biopsycho.2011.05.003 [doi]

Wilkus, R. J., & Dodrill, C. B. (1989). Factors affecting the outcome of MMPI and neuropsychological assessments of psychogenic and epileptic seizure patients. *Epilepsia*, 30(3), 339-347.

Wilkus, R. J., Dodrill, C. B., & Thompson, P. M. (1984). Intensive EEG monitoring and psychological studies of patients with pseudoepileptic seizures. *Epilepsia*, 25(1), 100-107.

Williams, J. M. G., Mathews, A., & MacLeod, C. (1996). The emotional Stroop task and psychopathology. *Psychological Bulletin*, 120(1), 3.

Williamson, D. J., Drane, D. L., Stroup, E. S., Wilensky, S., Holmes, M. D., & Miller, J. W. (2005). Recent seizures may distort the validity of neurocognitive test scores in patients with epilepsy. *Epilepsia*, 46, 74-74.

Williamson, D. J., Holsman, M., Chaytor, N., Miller, J. W., & Drane, D. L. (2012). Abuse, not financial incentive, predicts non-credible cognitive performance in patients with psychogenic non-epileptic seizures. *The Clinical Neuropsychologist*, 26(4), 588-598.

Willig, C. (2013). *Introducing qualitative research in psychology*. Berkshire: McGraw-Hill International.

Wingenfeld, K., Riedesel, K., Petrovic, Z., Philippsen, C., Meyer, B., Rose, M., . . . Spitzer, C. (2011). Impact of childhood trauma, alexithymia, dissociation, and emotion suppression on emotional Stroop task. *Journal of Psychosomatic Research*, 70(1), 53-58.

Wingenfeld, K., Rullkoetter, N., Mensebach, C., Beblo, T., Mertens, M., Kreisel, S., . . . Woermann, F. G. (2009). Neural correlates of the individual emotional Stroop in borderline personality disorder. *Psychoneuroendocrinology*, 34(4), 571-586.

Witgert, M. E., Wheless, J. W., & Breier, J. I. (2005). Frequency of panic symptoms in psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 6(2), 174-178.  
doi:10.1016/j.yebeh.2004.11.005

Wolf, L. D., Hentz, J. G., Ziemba, K. S., Kirlin, K. A., Noe, K. H., Hoerth, M. T., . . . Locke, D. E. C. (2015). Quality of life in psychogenic nonepileptic seizures and epilepsy: The role of somatization and alexithymia. *Epilepsy & Behavior*, 43(0), 81-88.  
doi:http://dx.doi.org/10.1016/j.yebeh.2014.12.010

Wood, B. L., McDaniel, S., Burchfiel, K., & Erba, G. (1998). Factors distinguishing families of patients with psychogenic seizures from families of patients with epilepsy. *Epilepsia*, 39(4), 432-437. doi:http://dx.doi.org/10.1111/j.1528-1157.1998.tb01396.x

World Health Organisation. (WHO; 1992). *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva: Switzerland: World Health Organisation.

Worsley, C., Whitehead, K., Kandler, R., & Reuber, M. (2011). Illness perceptions of healthcare workers in relation to epileptic and psychogenic nonepileptic seizures (vol 20, pg 668, 2011). *Epilepsy & Behavior*, 21(4), 495-495. doi:10.1016/j.yebeh.2011.04.065

Wright, S. P. (1992). Adjusted p-values for simultaneous inference. *Biometrics*, 48, 1005-1013.

Wyatt, C., Laraway, A., & Weatherhead, S. (2014). The experience of adjusting to a diagnosis of non-epileptic attack disorder (NEAD) and the subsequent process of psychological therapy. *Seizure*, 23(9), 799-807.  
doi:http://dx.doi.org/10.1016/j.seizure.2014.06.012

Xu, P., Xiong, X., Xue, Q., Li, P., Zhang, R., Wang, Z., . . . Yao, D. (2014). Differentiating between psychogenic nonepileptic seizures and epilepsy based on common spatial pattern of weighted EEG resting networks. *IEEE Transactions on Bio-Medical Engineering*, 61(6), 1747-1755. doi:10.1109/TBME.2014.2305159 [doi]

Xue, Q., Wang, Z. -, Xiong, X. -, Tian, C. -, Wang, Y. -, & Xu, P. (2013). Altered brain connectivity in patients with psychogenic non-epileptic seizures: A scalp electroencephalography study. *Journal of International Medical Research*, 41(5), 1682-1690. doi:http://dx.doi.org/10.1177/0300060513496170

Yi, Y. Y., Kim, H. D., Lee, J. S., Cheon, K. A., & Kang, H. C. (2014). Psychological problems and clinical outcomes of children with psychogenic non-epileptic seizures. *Yonsei Medical Journal*, 55(6), 1556-1561. doi:10.3349/ymj.2014.55.6.1556 [doi]

Yilmaz, U., Serdaroglu, A., Gurkas, E., Hirfanoglu, T., & Cansu, A. (2013). Childhood paroxysmal nonepileptic events. *Epilepsy & Behavior*, 27(1), 124-129.  
doi:10.1016/j.yebeh.2012.12.028

Zaidi, A., Crampton, S., Clough, P., Fitzpatrick, A., & Scheepers, B. (1999). Head-up tilting is a useful provocative test for psychogenic non-epileptic seizures. *Seizure*, 8(6), 353-355. doi:10.1053/seiz.1999.0302

Zajonc, R. B. (1980). Feeling and thinking: Preferences need no inferences. *American Psychologist*, 35(2), 151.

Zalsman, G., & Dror, S. (2002). Hypnosis provoked pseudoseizures: A case report and literature review. *American Journal of Clinical Hypnosis*, 45(1), 47-53.

Zaroff, C. M., Myers, L., Barr, W. B., Luciano, D., & Devinsky, O. (2004). Group psychoeducation as treatment for psychological nonepileptic seizures. *Epilepsy & Behavior*, 5(4), 587-592. doi:10.1016/j.yebeh.2004.03.005

Zhang, Y. C., Bromfield, E. B., Hurwitz, S., Nelson, A., Sylvia, K., & Dworetzky, B. A. (2009). Comparison of outcomes of video/EEG monitoring between patients with epileptic seizures and those with psychogenic nonepileptic seizures. *Epilepsy & Behavior*, 15(3), 303-307. doi:10.1016/j.yebeh.2009.04.008; 10.1016/j.yebeh.2009.04.008

Zigmond, A. S., & Snaith, R. P. (1983). The Hospital Anxiety and Depression Scale. *Acta Psychiatrica Scandinavica*, 67(6), 361-370.

## **Appendix I. Confirmation of ethical approval (quantitative)**



### **National Research Ethics Service**

**The Joint South London and Maudsley and the Institute of Psychiatry NHS  
Research Ethics Committee**

South London REC Office (2)  
1st Floor, Camberwell Building  
94 Denmark Hill  
London  
SE5 9RS

Telephone: 020 3299 5033

17 December 2008

Miss Susannah Pick  
PhD Student  
PO Box 78 Department of Psychology  
Institute of Psychiatry  
De Crespigny Park  
London SE5 8AF

Dear Miss Pick

**Full title of study:** An experimental investigation of aspects of emotional processing in patients diagnosed with Dissociative Seizures  
**REC reference number:** 08/H0807/82

Thank you for your letter of 20 November 2008, responding to the Committee's request for further information on the above research and submitting revised documentation.

The further information has been considered on behalf of the Committee by the Chair.

#### **Confirmation of ethical opinion**

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation as revised, subject to the conditions specified below.

#### **Ethical review of research sites**

The Committee has designated this study as exempt from site-specific assessment (SSA). The favourable opinion for the study applies to all sites involved in the research. There is no requirement for other Local Research Ethics Committees to be informed or SSA to be carried out at each site.

#### **Conditions of the favourable opinion**

The favourable opinion is subject to the following conditions being met prior to the start of the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

Management permission at NHS sites ("R&D approval") should be obtained from the relevant care organisation(s) in accordance with NHS research governance arrangements.

This Research Ethics Committee is an advisory committee to London Strategic Health Authority  
The National Research Ethics Service (NRES) represents the NRES Directorate within  
the National Patient Safety Agency and Research Ethics Committees in England



Guidance on applying for NHS permission is available in the Integrated Research Application System or at <http://www.rdforum.nhs.uk>.

### Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

| <i>Document</i>                                                           | <i>Version</i>  | <i>Date</i>       |
|---------------------------------------------------------------------------|-----------------|-------------------|
| GP letter                                                                 | 1.0             | 01 August 2008    |
| Healthy control assessment schedule                                       | 1.0             | 01 August 2008    |
| Patient assessment schedule                                               | 1.0             | 01 August 2008    |
| Healthy control invitation letter                                         | 1.0             | 01 August 2008    |
| Patient invitation letter                                                 | 1.1             | 01 August 2008    |
| Covering Letter                                                           |                 | 11 September 2008 |
| Protocol                                                                  | 1               | 01 August 2008    |
| Application                                                               | 1               | 26 September 2008 |
| CV Dr L Goldstein                                                         |                 | 24 July 2008      |
| Examples of IAP task stimuli                                              | 1.0             | 01 August 2008    |
| Circular email: Healthy Controls                                          | 1.0             | 01 August 2008    |
| Recruitment leaflet: Healthy Controls                                     | 1.0             | 01 August 2008    |
| Questionnaire: Additional information on questionnaire measures           | 1.0             | 01 September 2008 |
| Compensation Arrangements                                                 | KCL             |                   |
| Peer Review                                                               | MPhil/PhD board | 29 July 2008      |
| Investigator CV                                                           | Susannah Pick   | 11 September 2008 |
| CV Dr J Mellors                                                           |                 | 24 July 2008      |
| Participant Consent Form: Participants without Dissociative Seizures      | 1.1             | 01 November 2008  |
| Participant Consent Form: Participants with Dissociative Seizures         | 1.1             | 01 November 2008  |
| Participant Information Sheet: Participants without Dissociative Seizures | 1.1             | 01 November 2008  |
| Participant Information Sheet: Participants with Dissociative Seizures    | 1.2             | 01 November 2008  |
| Letter of invitation to participant                                       | 1.2             | 01 November 2008  |
| Response to Request for Further Information                               | 1               | 20 November 2008  |

### Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

### After ethical review

Now that you have completed the application process please visit the National Research Ethics Website > After Review

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

The attached document "After ethical review – guidance for researchers" gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Progress and safety reports
- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

We would also like to inform you that we consult regularly with stakeholders to improve our service. If you would like to join our Reference Group please email [referencegroup@nres.npsa.nhs.uk](mailto:referencegroup@nres.npsa.nhs.uk).

**08/H0807/82****Please quote this number on all correspondence**

With the Committee's best wishes for the success of this project

Yours sincerely



**Mr T Eaton**  
**Chair**

Email: [ethics.office@iop.kcl.ac.uk](mailto:ethics.office@iop.kcl.ac.uk)

*Enclosures:* "After ethical review – guidance for researchers"

*Copy to:* Mrs Gill Lambert, SLAM R&D

## **Appendix 2. Confirmation of ethical approval (qualitative)**



### **National Research Ethics Service** **South East London Research Ethics Committee (REC) 4**

(Formerly known as The Joint South London and Maudsley and Institute of Psychiatry Research Ethics Committee)

South London REC Office (2)  
1st Floor, Camberwell Building  
94 Denmark Hill  
London  
SE5 9RS

Tel: 020 3299 5033  
Fax: 020 3299 5085

08 June 2010

Miss Susannah Pick  
PhD Student  
Department of Psychology, PO Box 78  
Institute of Psychiatry, KCL  
De Crespigny Park  
London  
SE5 8AF

Dear Miss Pick

**Study title:** An experimental investigation of aspects of emotional processing in patients diagnosed with Dissociative Seizures  
**REC reference:** 08/H0807/82  
**Protocol number:**  
**Amendment number:** 2  
**Amendment date:** 26 April 2010

The above amendment was reviewed by the Sub-Committee in correspondence.

#### **Ethical opinion**

Favourable Opinion

The members of the Committee taking part in the review gave a favourable ethical opinion of the amendment on the basis described in the notice of amendment form and supporting documentation.

#### **Approved documents**

The documents reviewed and approved at the meeting were:

| Document                                                                   | Version | Date          |
|----------------------------------------------------------------------------|---------|---------------|
| Protocol                                                                   | 1.1     | 26 April 2010 |
| Notice of Substantial Amendment (non-CTIMPs)                               | 2       | 26 April 2010 |
| Covering Letter                                                            |         | 26 April 2010 |
| Questionnaire: Qualitative Interview Schedule                              | 1.0     | 26 April 2010 |
| Participant Consent Form: People with Dissociative Seizures                | 1.0     | 26 April 2010 |
| Participant Information Sheet: Dissociative Seizures: Follow Up Interviews | 1.0     | 26 April 2010 |

This Research Ethics Committee is an advisory committee to London Strategic Health Authority  
The National Research Ethics Service (NRES) represents the NRES Directorate within  
the National Patient Safety Agency and Research Ethics Committees in England

#### **Membership of the Committee**

The members of the Committee who took part in the review are listed on the attached sheet.

#### **R&D approval**

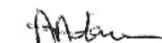
All investigators and research collaborators in the NHS should notify the R&D office for the relevant NHS care organisation of this amendment and check whether it affects R&D approval of the research.

#### **Statement of compliance**

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

|                                                             |
|-------------------------------------------------------------|
| Please quote this number on all correspondence: 08/H0807/82 |
|-------------------------------------------------------------|

Yours sincerely



**Audrey Adams**  
**Committee Co-ordinator**

E-mail: [audrey.adams@nhs.net](mailto:audrey.adams@nhs.net)

Enclosures: *List of names and professions of members who took part in the review*

Copy to: *Mrs Gill Lambert*  
*R&D office for NHS care organisation at lead site*

### **Appendix 3. Information sheet for patients (quantitative)**

**Institute of  
Psychiatry**

**at The Maudsley**



## **Information Sheet for Participants with Dissociative Seizures**

You are invited to take part in a research study at the Institute of Psychiatry (IoP), King's College London. This study is part of a student research project (PhD) being undertaken in the Department of Psychology, IoP. Before you decide whether to participate, you need to understand why the research is being done and what it would involve for you. Please take time to read the following information carefully. Talk to others about the study if you wish. Feel free to ask us if there is anything that is not clear or if you would like more information.

**Study title: Emotional processing in patients diagnosed with Dissociative Seizures. (LREC: 08/H0807/82)**

**Purpose of the research:** The aim of this study is to look at how people diagnosed with Dissociative Seizures perceive and respond to emotionally meaningful information, in comparison to people without this diagnosis. Important differences in how emotional information is experienced and responded to may contribute to some of the symptoms that occur in Dissociative Seizures, and therefore this investigation may provide a more detailed understanding of the condition. People diagnosed with Dissociative Seizures vary widely in many ways. Therefore this research will also assess whether different ways of perceiving emotional information are linked to other characteristics, such as personality traits, particular life events, and seizure-symptoms. We will examine how these characteristics differ from participants who do not have this diagnosis.

**Why have I been invited?** You have been invited to participate on the basis of your recent visit to the Neuropsychiatry Unit at the Maudsley Hospital or the Psychological Medicine Department at King's College Hospital. A total of 40 people diagnosed with Dissociative Seizures and 40 people without this diagnosis will be included in the study.

**Do I have to take part?** No. It is entirely your decision whether you take part in the study or not. If you are interested in taking part, you will be given a copy of this information sheet

and asked to sign a form agreeing to take part. However, you would be able to withdraw from the study at any time, or refrain from particular aspects of the research without giving a reason. If you decide not to participate or you withdraw from the study at a later stage, this will in no way affect the medical treatment that you receive now or in the future.

**What will happen to me if I decide to take part?** First, the researcher (Susannah Pick) would need to assess your eligibility for the study on the basis of information about you that was collected when you were seen at the clinic. Following this, the researcher would let you know if you are eligible for the study. If you are not eligible you won't need to do anything else. If you are eligible, you would be invited to the Institute of Psychiatry (adjacent to the Maudsley Hospital, Denmark Hill) on a day that is convenient for you. On arrival, any questions will be answered and the procedure for the study will be explained in full. You will be provided with an a sheet which includes details of each test and questionnaire you will be asked to complete. You will also be able to ask any questions about the tests at this stage.

When you are ready to begin, you would initially carry out three computerised tasks that involve looking at a variety of pictures and making simple judgements about them, such as whether they are pleasant or unpleasant. During two of these tasks, small sensors will be attached to three fingers on one of your hands (these sensors do not lead to any discomfort). You are not required to have previous experience with computers for these tasks. Four brief tasks will then be administered by the researcher, and finally you would be asked to complete a number of questionnaires. The researcher will be present throughout the session, to answer any questions or assist you as necessary. The study will involve only one visit to the IoP although it will take approximately 5 hours to complete. However, several breaks will be provided (including a lunch break).

**Expenses & payment:** You will receive a payment of £30 as reimbursement for your time and up to £20 for your travel expenses incurred in getting to the IOP.

**Possible risks & disadvantages of taking part:** No direct health risks are expected to result from this study. However, it is important for you to be aware that some of the pictures to be presented to you include unpleasant content, and therefore may be distressing. However they are generally no more distressing than images commonly seen on the television news or in newspapers.

In addition, some of the questionnaires given to you include questions about potentially sensitive issues that some participants may find difficult, such as whether or not you have experienced any traumatic events in your lifetime, and the nature of these events. It is important for you to be aware that you are not obliged to complete these questionnaires,

and you will be given the option to opt-out of completing these on the day. If you feel concerned about these aspects of the study please feel free to discuss this further with the researcher. If you feel that any aspect of the research has caused you to be distressed on the day, you would be welcome to talk to the researcher about this, who will be available to provide any information, support and reassurance you may need. We can also provide you with a range of options to support you in managing any distress, and would discuss these with you if necessary to determine the most appropriate for you at the time.

Due to the length of the testing session and the number of different activities involved, it is also possible that you may feel tired by the end of the day. For this reason, several breaks will be included and the tasks that require most concentration and attention will be completed earlier in the day. Additional breaks can also be taken on request.

**Potential benefits of taking part:** There will be no immediate benefit to you for taking part in this study. However the aim of the research is to increase the current understanding of this condition, which would indirectly benefit both yourself and other people who are diagnosed with DS.

**What happens if something goes wrong?** While we do not anticipate any adverse effects from taking part in this study, if you are harmed by taking part in this research project, there are no special compensation arrangements. If you are harmed due to someone's negligence you may have grounds for a legal action, but you may have to pay for it. Regardless of this, if you wish to complain, or have any concerns about any aspect of how you have been treated or approached during the course of the study, the normal National Health Service complaints mechanisms should be available to you. Furthermore, if you have any problems or queries during the course of the study, you would be welcome to address these with the researcher.

**Will my taking part be kept confidential?** All information provided will be treated as confidential and will be managed exclusively by the researchers involved. You will not be identified on our computers or in publications by name. Participants will be given a unique reference number for storage and analysis of their data. Therefore this is anonymous after the point of collection. Any information about you will have your name and address removed so that you cannot be recognised from it. This data will be stored on password-protected computers at the IoP for 10 years after which it will be destroyed. However there are some limitations to the confidentiality of the data collected during the study. If there is any indication of risk to your own/another's safety, the information will be passed on to other relevant professionals. In this event, you would be informed in advance.

**What will happen to the results of the study:** The results of the study will be summarised in articles that will be submitted for publication in scientific journals and presented at conferences. You will not be identified in any published report or article unless your consent is sought to do so. Copies of any published articles and a brief summary of the results will be sent to participants on request. However each individual person's results will not be provided. The results of this study will also form the basis of a doctoral thesis (PhD) that will be written by the primary researcher (Susannah Pick).

**Discontinuation of the study by the investigators:** At any time during the study, the investigators have the right to discontinue your participation in the study for any reason. If later on in the study it is concluded that you are no longer able to consent to participating, we would like to be able to continue to use any data that we have already collected, although this would not include any of personal details that could identify you in any way.

**Who is organising the research?** The study is being funded by the Department of Psychology, IoP. It is being organised by Miss Susannah Pick (Postgraduate Research Student), under the supervision of Professor Laura Goldstein (Professor of Clinical Neuropsychology, IOP) and Dr John Mellers (Consultant Neuropsychiatrist, SLaM NHS Foundation Trust).

**Who has reviewed the study?** This study has been granted ethical approval by the Joint South London and Maudsley & Institute of Psychiatry NHS Research Ethics Committee (Ref: 08/H0807/82). The study has also been reviewed by the Dept of Psychology PhD Research Committee.

**Further information & contact details:** If you wish to discuss any aspect of the study please feel free to contact the researcher, referring to the below contact details.

***Susannah Pick***

*Department of Psychology, PO Box 78*

*Institute of Psychiatry*

*King's College London*

*De Crespigny Park*

*London*

*SE5 8AF*

*Tel: 020 7848 0766*

*Email: [susannah.pick@kcl.ac.uk](mailto:susannah.pick@kcl.ac.uk)*

If you require more information about the study, you can also contact Professor Laura Goldstein (Professor of Clinical Neuropsychology) Tel 020 7848 0218



([I.goldstein@kcl.ac.uk](mailto:I.goldstein@kcl.ac.uk)) or Dr John Mellers (Consultant Neuropsychiatrist) Tel 020 3228 2330.

If you wish to seek independent advice on whether to participate or not you can contact the Patient Advice & Liaison Service on 0800 731 2864 or [PALS@slam.nhs.uk](mailto:PALS@slam.nhs.uk).

Thank you for reading this information sheet. You can keep this copy. If you have understood the contents of this sheet and wish to take part, please inform the researcher that you are willing to sign the consent form. If you have any further questions, please feel free to ask them now.

## **Appendix 4. Information sheet for control participants (quantitative)**

**Institute of  
Psychiatry**

**at The Maudsley**

**KING'S**  
College  
**LONDON**  
*Founded 1829*  
University of London

### **Information Sheet for People without Dissociative Seizures**

You are invited to take part in a research study at the Institute of Psychiatry (IoP), King's College London. This study is part of a student research project (PhD) being undertaken in the Department of Psychology, IOP. Before you decide whether to participate, you need to understand why the research is being done and what it would involve for you. Please take time to read the following information carefully. Talk to others about the study if you wish. Feel free to ask us if there is anything that is not clear or if you would like more information.

**Study title: Emotional processing in patients diagnosed with Dissociative Seizures.**  
(LREC: 08/H0807/82)

**Purpose of the research:** Dissociative Seizures are episodes of altered awareness and behaviour that resemble epilepsy, but are not found to have a physical cause. It is thought instead that Dissociative Seizures are caused by psychological factors. The aim of this study is to look at how people diagnosed with Dissociative Seizures perceive and respond to emotionally meaningful information, in comparison to people without this diagnosis. Important differences in how emotional information is experienced and responded to may contribute to some of the symptoms that occur in Dissociative Seizures, and therefore this investigation may provide a more detailed understanding of the condition. People diagnosed with Dissociative Seizures vary widely in many ways. Therefore this research will also assess whether different ways of perceiving emotional information are linked to other characteristics, such as personality traits, particular life events, and seizure-symptoms. We will examine how these characteristics differ from participants who do not have this diagnosis.

**Why have I been invited?** You have been invited to participate on the basis of your response to our recent advertisement/having registered your details on the MindSearch database. A total of 40 people diagnosed with Dissociative Seizures and 40 people without this diagnosis will be included in the study. We would still like to ask you some further questions about your medical history to determine if you are a suitable candidate for the study.

**Do I have to take part?** No. It is entirely your decision whether you take part in the study or not. If you decide to take part, you will be given a copy of this information sheet to keep and asked to sign a form agreeing to take part. However, you would be able to withdraw from the study at any time or refrain from particular aspects of the research if you so wish, without giving a reason.

**What will happen to me if I decide to take part?** You would be invited to the Institute of Psychiatry (adjacent to the Maudsley Hospital, Denmark Hill) to take part in the study on a day that is convenient for you. On arrival, any questions will be answered and the procedure for the study will be explained in full by the researcher. You will be provided with a sheet which includes details of each test and questionnaire you will be asked to complete. You will also be able to ask any questions about the tests at this stage.

When you are ready to begin, you will carry out three computerised tasks that involve looking at a variety of pictures and making simple judgements about them, such as whether they are pleasant or unpleasant. During two of these tasks, small sensors will be attached to three fingers on one of your hands (these sensors do not lead to any discomfort). You are not required to have previous experience with computers for these tasks. Four brief tasks will also be administered by the researcher, and you will also be asked to complete a number of questionnaires. The researcher will be present throughout the session, to answer any questions or assist you as necessary. The study will involve only one visit to the IoP although it will take approximately 5 hours to complete. However, several breaks will be provided (including a lunch break).

**Expenses & payment:** You will receive a payment of £30 as reimbursement for your time and up to £20 for your travel expenses incurred in getting to and from the IoP. **We will ask you to provide receipts or car mileage details so we can reimburse up to £20 for the travel expenses.**

**Possible risks & disadvantages of taking part:** No direct health risks are expected to result from this study. However, it is important for you to be aware that some of the pictures to be presented to you include unpleasant content, and therefore may be distressing. However they are generally no more distressing than images commonly seen on the television news or in newspapers.

In addition, some of the questionnaires given to you include questions about potentially sensitive issues that some participants may find difficult, such as whether or not you have experienced any traumatic events in your lifetime, and the nature of these events. It is

important for you to be aware that you are not obliged to complete these questionnaires, and you will be given the option to opt-out of completing these on the day. If you feel concerned about these aspects of the study please feel free to discuss this further with the researcher. If you feel that any aspect of the research has caused you to be distressed on the day, you would be welcome to talk to the researcher about this, who will be available to provide any information, support and reassurance you may need. We can also provide you with a range of options to support you in managing any distress, and would discuss these with you if necessary to determine the most appropriate for you at the time.

Due to the length of the testing session and the number of different activities involved, it is also possible that you may feel tired by the end of the day. For this reason, several breaks will be included and the tasks that require most concentration and attention will be completed earlier in the day. Additional breaks can also be taken on request.

**Possible benefits of taking part:** There will be no immediate benefit to you for taking part in this study. However the aim of the research is to increase the current understanding of Dissociative Seizures and could help people diagnosed with that condition.

**What happens if something goes wrong?** While we do not anticipate any adverse effects from taking part in this study, if you are harmed by taking part in this research project, there are no special compensation arrangements. If you are harmed due to someone's negligence you may have grounds for a legal action, but you may have to pay for it. Regardless of this, if you wish to complain, or have any concerns about any aspect of how you have been treated or approached during the course of the study, the normal National Health Service complaints mechanisms should be available to you. Furthermore, if you have any problems or queries during the course of the study, you would be welcome to address these with the researcher.

**Will my taking part be kept confidential?** All information provided will be treated as confidential and will be managed exclusively by the researchers involved. You will not be identified on our computers or in publications by name. Participants will be given a unique reference number for storage and analysis of their data. Therefore this is anonymous after the point of collection. Any information about you will have your name and address removed so that you cannot be recognised from it. These data will be stored on password-protected computers at the IoP for 10 years after which it will be destroyed. However there are some limitations to the confidentiality of the data collected during the study. If there is any indication of risk to your own/another's safety, the information will be passed on to other relevant professionals. In this event, you would be informed in advance.

**What will happen to the results of the study?** The results of the study will be summarised in articles that will be submitted for publication in scientific journals and presented at conferences. You will not be identified in any published report or article unless your consent is sought to do so. Copies of any published articles and a brief summary of the results will be sent to participants on request. However each individual person's results will not be provided. The findings of this study will also form the basis of a doctoral thesis (PhD) that will be written by the primary researcher (Susannah Pick).

**Discontinuation of the study by the investigators:** At any time during the study, the investigators have the right to discontinue your participation in the study for any reason. If later on in the study it is concluded that you are no longer able to consent to participating, we would like to be able to continue to use any data that we have already collected, although this would not include any of personal details that could identify you in any way.

**Who is organising the research?** The study is being funded by the Department of Psychology, IoP. It is being organised by Miss Susannah Pick (Postgraduate Research Student), under the supervision of Professor Laura Goldstein (Professor of Clinical Neuropsychology, IoP) and Dr John Mellers (Consultant Neuropsychiatrist, SLaM NHS Foundation Trust).

**Who has reviewed the study?** This study has been granted ethical approval by the Joint South London and Maudsley & Institute of Psychiatry NHS Research Ethics Committee (Ref: 08/H0807/82). The study has also been reviewed by the Dept of Psychology PhD Research Committee.

**Further information & contact details:** If you wish to discuss any aspect of the study please feel free to contact the researcher, referring to the below contact details.

***Susannah Pick***

*Department of Psychology, PO Box 78*

*Institute of Psychiatry*

*King's College London*

*De Crespigny Park*

*London*

*SE5 8AF*

*Tel: 020 7848 0766*

*Email: [susannah.pick@iop.kcl.ac.uk](mailto:susannah.pick@iop.kcl.ac.uk)*

If you require more information about the study, you can also contact Professor Laura Goldstein (Professor of Clinical Neuropsychology) Tel 020 7848 0218

([l.goldstein@iop.kcl.ac.uk](mailto:l.goldstein@iop.kcl.ac.uk)) or Dr John Mellers (Consultant Neuropsychiatrist) Tel 020 3228 2330.

Thank you for reading this information sheet. You can keep this copy. If you have understood the contents of this sheet and wish to take part, please inform the researcher that you are willing to sign the consent form. If you have any further questions, please feel free to ask them now.

## **Appendix 5. Consent form for patients (quantitative)**

**Institute of  
Psychiatry**

**at The Maudsley**



### **Consent Form for People with Dissociative Seizures**

Emotional Processing in Patients Diagnosed with Dissociative Seizures  
(LREC: 08/H0807/82)

Researcher: Miss Susannah Pick

**Please tick each box if you agree to the statement**

☐

I have read the information sheet for the above stated study, have had time to consider the information and have had the opportunity to ask questions.

☐

Further, I understand that I may seek information about each test either before or after it is given.

☐

I understand that my participation is voluntary and I am free to withdraw from the testing at any time without giving a reason, and without my current / future medical care being affected.

☐

I understand that my personal information will be kept confidential.

☐

I consent to members of the research team for this study, who are from the Institute of Psychiatry and the Maudsley Hospital, having access to my medical records, when this is relevant to my taking part in the research.

☐

I consent that in the event of a future loss in capacity, any data already collected may continue to be used, confidentially, in connection with this study

☐

I would like to receive a summary of the results of the study

☐

I agree to take part in the study

\_\_\_\_\_  
Signature of participant

\_\_\_\_\_  
Date

\_\_\_\_\_  
Name of participant (in capitals)



I have explained the study to (name of participant) and have answered questions honestly and fully.

\_\_\_\_\_  
Signature of investigator

\_\_\_\_\_  
Date

\_\_\_\_\_  
Name of investigator (in capitals)



## **Appendix 6. Consent form for control participants (quantitative)**

**Institute of  
Psychiatry**

**at The Maudsley**



### **Consent Form for People without Dissociative Seizures**

Emotional Processing in Patients Diagnosed with Dissociative Seizures  
(LREC: 08/H0807/82)

Researcher: Miss Susannah Pick

**Please tick each box if you agree to the statement**

☐ I have read the information sheet for the above stated study, have had time to consider the information and have had the opportunity to ask questions.

☐ Further, I understand that I may seek information about each test either before or after it is given.

☐ I understand that my participation is voluntary and I am free to withdraw from the testing at any time without giving a reason.

☐ I understand that my personal information will be kept confidential.

☐ I consent that in the event of a future loss in capacity, any data already collected may continue to be used, confidentially, in connection with this study

☐ I would like to receive a summary of the results of the study

☐ I agree to take part in the study

\_\_\_\_\_  
Signature of participant

\_\_\_\_\_  
Date

\_\_\_\_\_  
Name of participant (in capitals)



I have explained the study to (name of participant) and have answered questions honestly and fully.

---

Signature of investigator

---

Date

---

Name of investigator (in capitals)

## **Appendix 7. Information sheet (qualitative)**

**Institute of  
Psychiatry**  
  
**at The Maudsley**

**KING'S**  
College  
**LONDON**  
*Founded 1829*  
University of London

### **Information Sheet for Participants with Dissociative Seizures: Follow-Up Interviews**

You are invited to take part in a research interview at the Institute of Psychiatry (IoP), King's College London. This study is part of a student research project (PhD) being undertaken in the Department of Psychology, IoP. Before you decide whether to participate, you need to understand why the research is being done and what it would involve for you. Please take time to read the following information carefully. Talk to others about the study if you wish. Feel free to ask us if there is anything that is not clear or if you would like more information.

**Study title: Emotional processing in patients diagnosed with Dissociative Seizures: Follow-Up Interviews (LREC: 08/H0807/82)**

**Purpose of the research:** The main aim of this study is to look at how people diagnosed with Dissociative Seizures perceive and respond to emotionally meaningful information, in comparison to people without this diagnosis. Important differences in how emotional information is experienced and responded to may contribute to some of the symptoms that occur in Dissociative Seizures, and therefore this investigation may provide a more detailed understanding of the condition.

We are now carrying out a follow-up study, to try to find out how patients diagnosed with Dissociative Seizures think that emotions and stress affect them, their lives, and their symptoms. This involves carrying out in-depth interviews with patients who have already taken part in the main study, focusing on these topics. We hope that this follow-up study will provide further insights into some possible causes of the disorder, and give us information about patients' own thoughts and feelings about this.

**Why have I been invited?** You have been invited to participate as you have now completed the main part of this study. We are now interested in finding out about your opinions on how emotions and stress influence your symptoms.

**Do I have to take part?** No. It is entirely your decision whether you take part in the study or not. If you are interested in taking part, you will be given a copy of this information sheet and asked to sign a form agreeing to take part. However, you would be able to withdraw from the study at any time, or refrain from particular aspects of the research without giving a reason. If you decide not to participate or you withdraw from the study at a later stage, this will in no way affect the medical treatment that you receive now or in the future.

**What will happen to me if I decide to take part?**

. If you agree to take part in this follow-up study we would agree that you would have one further meeting with the researcher (SP), during which you would be asked questions about your views and understanding of how emotions and stress affect you and your seizures. The researcher will guide the interview with some specific questions, but you will also be free to discuss issues that you feel are important to the subject. This interview would take around 30-60 minutes, and would be recorded using a digital voice recorder for later analysis.

**Expenses & payment:** You will receive a payment of £15 as reimbursement for your time and up to £20 for your travel expenses incurred in getting to the IOP. We will ask you to provide receipts or car mileage details so we can reimburse up to £20 for the travel expenses.

**Possible risks & disadvantages of taking part:** No direct health risks are expected to result from this study. However, some of the questions asked during the interview may pertain to potentially sensitive issues that you may find difficult or uncomfortable, such as how emotionally upsetting events/situations tend to affect you.

It is important for you to be aware that you are not obliged to answer all of the questions, and you will be given the option to not answer or move to the next question if you wish, or to withdraw from the interview at any time. If you feel concerned about these aspects of the study please feel free to discuss this further with the researcher.

If you feel that any aspect of the research has caused you to be distressed on the day, you would be welcome to talk to the researcher about this, who will be available to provide any information, support and reassurance you may need. We can also provide you with a range of options to support you in managing any distress, and would discuss these with you if necessary to determine the most appropriate for you at the time.

**Potential benefits of taking part:** There will be no immediate benefit to you for taking part in this study. However, this follow-up study would give you the opportunity to express

your own views about how emotion/stress contributes to your symptoms. We hope this will enhance understanding of this disorder among professionals and the general public alike, which would indirectly benefit you and other people diagnosed with Dissociative Seizures.

**What happens if something goes wrong?** While we do not anticipate any adverse effects from taking part in this study, if you are harmed by taking part in this research project, there are no special compensation arrangements. If you are harmed due to someone's negligence you may have grounds for a legal action, but you may have to pay for it. Regardless of this, if you wish to complain, or have any concerns about any aspect of how you have been treated or approached during the course of the study, the normal National Health Service complaints mechanisms should be available to you. Furthermore, if you have any problems or queries during the course of the study, you would be welcome to address these with the researcher.

**Will my taking part be kept confidential?** All information provided will be treated as confidential and will be managed exclusively by the researchers involved. You will not be identified on our computers or in publications by name. Participants will be given a unique reference number for storage and analysis of their data. Therefore this is anonymous after the point of collection. Any information about you will have your name and address removed so that you cannot be recognised from it. This data will be stored on password-protected computers at the IoP for 10 years after which it will be destroyed. The recordings that we make of the interviews will be kept securely until they have been typed out (transcribed) and then they will be safely disposed of to preserve your anonymity.

However there are some limitations to the confidentiality of the data collected during the study. If there is any indication of risk to your own/another's safety, the information will be passed on to other relevant professionals. In this event, you would be informed in advance.

**What will happen to the results of the study:** The results of the study will be summarised in articles that will be submitted for publication in scientific journals and presented at conferences. You will not be identified in any published report or article unless your consent is sought to do so. Copies of any published articles and a brief summary of the results will be sent to participants on request. However each individual person's results will not be provided. The results of this study will also form the basis of a doctoral thesis (PhD) that will be written by the primary researcher (Susannah Pick).

**Discontinuation of the study by the investigators:** At any time during the study, the investigators have the right to discontinue your participation in the study for any reason. If

later on in the study it is concluded that you are no longer able to consent to participating, we would like to be able to continue to use any data that we have already collected, although this would not include any of personal details that could identify you in any way.

**Who is organising the research?** The study is being funded by the Department of Psychology, IoP. It is being organised by Ms Susannah Pick (Postgraduate Research Student), under the supervision of Professor Laura Goldstein (Professor of Clinical Neuropsychology, IOP) and Dr John Mellers (Consultant Neuropsychiatrist, SLAM NHS Foundation Trust).

**Who has reviewed the study?** This study has been granted ethical approval by the Joint South London and Maudsley & Institute of Psychiatry NHS Research Ethics Committee (Ref: 08/H0807/82). The study has also been reviewed by the Dept of Psychology PhD Research Committee.

**Further information & contact details:** If you wish to discuss any aspect of the study please feel free to contact the researcher, referring to the below contact details.

***Susannah Pick***

*Department of Psychology, PO Box 78*

*Institute of Psychiatry*

*King's College London*

*De Crespigny Park*

*London*

*SE5 8AF*

*Tel: 020 7848 0766*

*Email: [susannah.pick@iop.kcl.ac.uk](mailto:susannah.pick@iop.kcl.ac.uk)*

If you require more information about the study, you can also contact Professor Laura Goldstein (Professor of Clinical Neuropsychology) Tel 020 7848 0218 ([l.goldstein@iop.kcl.ac.uk](mailto:l.goldstein@iop.kcl.ac.uk)) or Dr John Mellers (Consultant Neuropsychiatrist) Tel 020 3228 2330.

If you wish to seek independent advice on whether to participate or not you can contact the Patient Advice & Liaison Service on 0800 731 2864 or [PALS@slam.nhs.uk](mailto:PALS@slam.nhs.uk).

Thank you for reading this information sheet. You can keep this copy. If you have understood the contents of this sheet and wish to take part, please inform the researcher that you are willing to sign the consent form. If you have any further questions, please feel free to ask them now.

## **Appendix 8. Consent form (qualitative)**

**Institute of  
Psychiatry**

**at The Maudsley**



### **Consent Form for People with Dissociative Seizures**

Emotional Processing in Patients Diagnosed with Dissociative Seizures:  
Follow-Up Interviews

(LREC: 08/H0807/82)

Researcher: Ms Susannah Pick

**Please tick each box if you agree to the statement**

☐

I have read the information sheet for the above stated study, have had time to consider the information and have had the opportunity to ask questions.

☐

Further, I understand that I may seek additional information about the follow-up study at any time.

☐

I understand that my participation is voluntary and I am free to withdraw from the testing at any time without giving a reason, and without my current / future medical care being affected.

☐

I understand that my personal information will be kept confidential.

☐

I consent to members of the research team for this study, who are from the Institute of Psychiatry and the Maudsley Hospital, having access to my medical records, when this is relevant to my taking part in the research.

☐

I consent that in the event of a future loss in capacity, any data already collected may continue to be used, confidentially, in connection with this study

☐

I consent to being interviewed where the interview will be recorded using electronic auditory recording equipment

☐

I consent to the interview being transcribed (typed out) in full, but that my identity may not be revealed in this transcript

☐

I consent to aspects of what I might say in the interview being quoted directly in reports about the study, as long as my identity is not revealed in such reports

☐ I would like to receive a summary of the results of the study

☐ I agree to take part in the study

\_\_\_\_\_  
Signature of participant

\_\_\_\_\_  
Date

\_\_\_\_\_  
Name of participant (in capitals)

☐ I have explained the study to (name of participant) and have answered questions honestly and fully.

\_\_\_\_\_  
Signature of investigator

\_\_\_\_\_  
Date

\_\_\_\_\_  
Name of investigator (in capitals)



## **Appendix 9. Ethical approval letter (amendment for travel reimbursement)**



**National Research Ethics Service**  
The Joint South London and Maudsley and The Institute of Psychiatry NHS  
Research Ethics Committee

South London REC Office (2)  
1st Floor, Camberwell Building  
94 Denmark Hill  
London  
SE5 9RS

24 August 2009

Miss Susannah Pick  
PhD Student  
Department of Psychology, PO Box 78  
Institute of Psychiatry, KCL  
De Crespigny Park  
London  
SE5 8AF

Dear Miss Pick

**Study title:** An experimental investigation of aspects of emotional processing in patients diagnosed with Dissociative Seizures  
**REC reference:** 08/H0807/82  
**Amendment number:** 3.1  
**Amendment date:** 08 July 2009

The above amendment was reviewed at the meeting of the Sub-Committee held on 21 August 2009.

### **Ethical opinion**

The members of the Committee taking part in the review gave a favourable ethical opinion of the amendment on the basis described in the notice of amendment form and supporting documentation.

### **Approved documents**

The documents reviewed and approved at the meeting were:

| Document                                                                        | Version | Date         |
|---------------------------------------------------------------------------------|---------|--------------|
| Participant Information Sheet: In patients diagnosed with dissociative seizures | 1.2     |              |
| Participant Information Sheet: In patients diagnosed with dissociative seizures | 1.1     |              |
| Participant Information Sheet: In patients diagnosed with dissociative seizures | 1.3     |              |
| Participant Information Sheet: In patients diagnosed with dissociative seizures | 1.2     |              |
| Notice of Substantial Amendment (non-CTIMPs)                                    | 3.1     | 08 July 2009 |
| Covering Letter                                                                 |         | 08 July 2009 |

This Research Ethics Committee is an advisory committee to London Strategic Health Authority  
The National Research Ethics Service (NRES) represents the NRES Directorate within  
the National Patient Safety Agency and Research Ethics Committees in England

### **R&D approval**

All investigators and research collaborators in the NHS should notify the R&D office for the relevant NHS care organisation of this amendment and check whether it affects R&D approval of the research.


### **Statement of compliance**

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

08/H0807/82:

Please quote this number on all correspondence

Yours sincerely

  
**Chris Ward**  
**Committee Co-ordinator**

E-mail: [chris.ward@kch.nhs.uk](mailto:chris.ward@kch.nhs.uk)

Copy to:

*Mrs Gill Lambert*  
*[R&D office for NHS care organisation at lead site]*

## **Appendix 10. Advertisement for healthy control participants**

**Institute of  
Psychiatry**

**at The Maudsley**

**KING'S**  
College  
**LONDON**  
*Founded 1829*  
University of London

# **Volunteers required for a Psychology research study**

## **Investigating subjective and physiological responses to emotionally arousing pictures**

We are seeking to recruit healthy volunteers to form a control group for a research study investigating the role of emotional processing biases in a specific clinical disorder. The study involves three computerised tasks. These involve making simple judgements about emotionally arousing pictures of people and things. Two of the tasks will also include recording of your responses to the items with small sensors on three of your fingers.

You will also be asked to complete several other tasks and questionnaires. The study will take approximately 3-4 hours to complete (including breaks), and you will be reimbursed £30 for your time and inconvenience.

You must be between 18 and 65 years old, have no history of / current psychiatric condition, neurological illness or substance dependence and be fluent in English. Participation in the research would be voluntary and you would be free to withdraw at any time.

If you are interested in taking part in the study or would like further information, please contact Susannah Pick on [susannah.pick@kcl.ac.uk](mailto:susannah.pick@kcl.ac.uk).

## **Appendix I I. Ictal symptoms questionnaire**

Participant Number

Date

### **ATTACK SYMPTOMS QUESTIONNAIRE**

**Please indicate whether you have experienced each of the following symptoms during your most recent attack and / or during your most severe attack by circling Y or N**

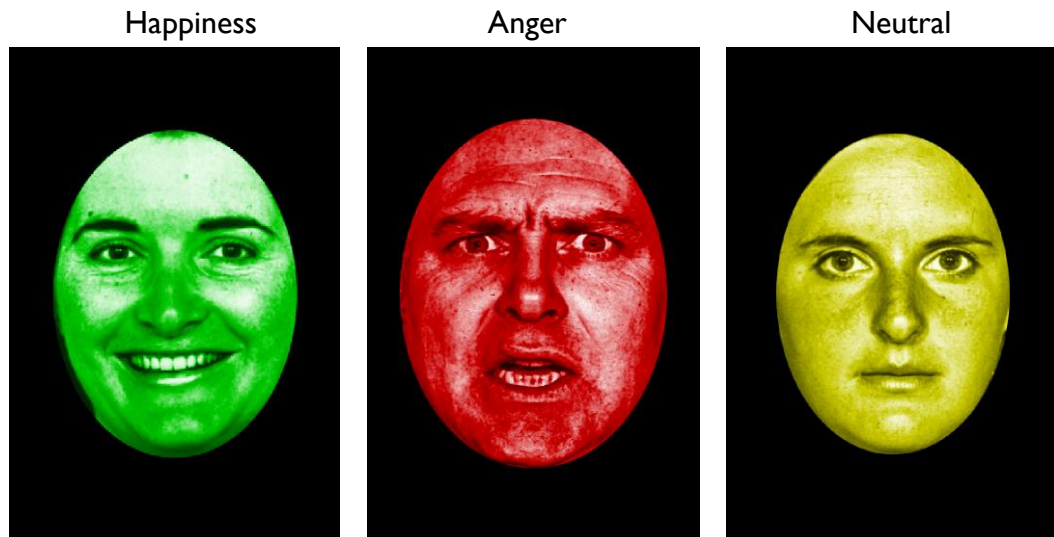
|                                                                      | Most recent attack |   | Most severe attack |   |
|----------------------------------------------------------------------|--------------------|---|--------------------|---|
| Shortness of breath or smothering sensation (CA)                     | Y                  | N | Y                  | N |
| Dizziness, unsteady feelings or faintness (MS)                       | Y                  | N | Y                  | N |
| Racing or pounding heart (AA)                                        | Y                  | N | Y                  | N |
| Trembling or shaking (AA)                                            | Y                  | N | Y                  | N |
| Sweating (AA)                                                        | Y                  | N | Y                  | N |
| Choking (CA)                                                         | Y                  | N | Y                  | N |
| Nausea or abdominal distress/<br>butterflies or knot in stomach (CA) | Y                  | N | Y                  | N |
| Numbness or tingling in arms, legs or face (GEN)                     | Y                  | N | Y                  | N |
| Dry mouth or throat (AA)                                             | Y                  | N | Y                  | N |
| Hot flushes or chills (GEN)                                          | Y                  | N | Y                  | N |
| Chest pains or discomfort (CA)                                       | Y                  | N | Y                  | N |
| Feeling that things are not real (MS)                                | Y                  | N | Y                  | N |
| Desire to escape from the scene of the attack (COG)                  | Y                  | N | Y                  | N |
| Thoughts or images that you cannot get rid of (COG)                  | Y                  | N | Y                  | N |

|                                                                                   |   |   |   |   |
|-----------------------------------------------------------------------------------|---|---|---|---|
| <b>Feelings of embarrassment (COG)</b>                                            | Y | N | Y | N |
| <b>Feeling that things around you are strange, unreal, foggy or detached (MS)</b> | Y | N | Y | N |
| <b>Feeling outside or detached from part or all of your body (MS)</b>             | Y | N | Y | N |
| <b>Feeling “cut-off” from your surroundings (MS)</b>                              | Y | N | Y | N |
| <b>Thinking you are going to die (COG)</b>                                        | Y | N | Y | N |
| <b>Thinking “I’m losing control” (COG)</b>                                        | Y | N | Y | N |
| <b>Thinking “I’m going crazy” (COG)</b>                                           | Y | N | Y | N |
| <b>Thinking “I’m going blind” (COG)</b>                                           | Y | N | Y | N |
| <b>Thinking “I’m going to be paralysed” (COG)</b>                                 | Y | N | Y | N |
| <b>Thinking “I’m going to scream” (COG)</b>                                       | Y | N | Y | N |
| <b>Thinking “My attack will never end” (COG)</b>                                  | Y | N | Y | N |
| <b>Thinking “I’m going to black out” (COG)</b>                                    | Y | N | Y | N |

CA = chest/abdomen symptoms; AA = autonomic arousal symptoms; COG = cognitive symptoms; MS = mental state symptoms; GEN = general seizure symptoms

## **Appendix 12. Stimuli used in the emotional Stroop test**

### **Example facial stimuli**



### **Full list of facial stimuli (from Ekman & Friesen, 1976)**

Experimental stimuli: 001, 003, 006, 014, 018, 021, 029, 030, 033, 034, 038, 041, 042, 044, 047, 048, 053, 056, 057, 061, 065, 074, 080, 083, 093, 096, 099, 101, 105, 110

### **Masking stimuli**



### **Appendix I3. Standardised instructions (emotional Stroop task)**

You are now going to be shown a pattern many times.

The pattern is an oval shape, made of several lines of colour.

The pattern will be shown in either red, yellow, or green,

(Please press space to continue)

Please say aloud the colour of the pattern, each time you see it.

Please say the colour as quickly and clearly as you can.

Remember: it is only the COLOUR of the pattern that you must look at, nothing more.

Press the space bar for some examples. When you have looked at each example,  
press space to continue.

Please look at the screen at all times during this task.

Also, try not to cough or make any other noises during this task, except saying the  
colour names.

Remember to stay as still as possible throughout the task.

(Please press space to continue)

Please say the colour of each pattern loudly and clearly.

You will now have the opportunity to practise.

Press space when you are ready to begin the practice items.

You have now completed the practice items.

Remember: it is very important that you do not make any other noises during this  
task, except for naming the colours.

When you say the colour names, please speak as loudly and clearly as you can, and  
answer as quickly but as accurately as possible.

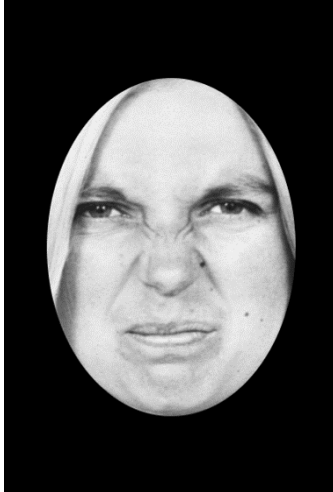
When you are ready to begin the task, please press space.

Thank you for completing this part of the research.

## **Appendix 14. Stimuli used in the facial expression processing task**

### **Example stimuli**

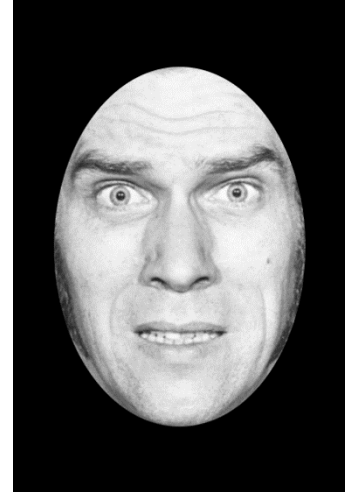
Disgust



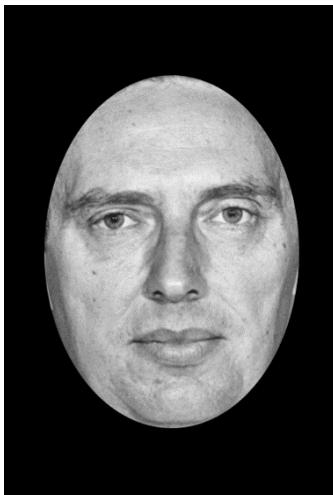
Happiness



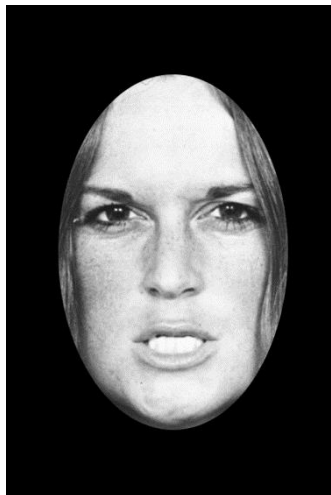
Fear



Neutral



Anger



### **Full list of facial stimuli (from Ekman & Friesen, 1976)**

Practice items: 022, 027, 028

Experimental items: 014, 016, 018, 020, 021, 034, 037, 038, 040, 041, 057, 059, 061, 064, 065, 085, 088, 089, 091, 092, 093, 095, 096, 098, 099, 101, 104, 105, 108, 110



## **Appendix 15. Standardised instructions (facial expression processing task)**

You will now see many different faces, showing various expressions.  
For each face, you will be asked to decide which emotion is expressed, and how intense you think it is.

Please answer as quickly but as accurately as you can.  
(Please press space to continue)

Some of the faces will show clear emotions, but others may seem neutral.  
Please report your immediate personal reaction to the facial expression, and no more.

There are no right or wrong answers.  
(Please press space to continue)

Before each face, you will see a white cross on the screen for some time.  
Next, the face will be shown for a few seconds.  
Then you will be asked to make two judgements about the facial expression you just saw.

(Please press space to continue)

First, you will be asked which of these emotions was in the face:

Happiness, anger, fear, disgust, neutral.

Please give your answer by pressing the number key that is shown next to the emotion you saw (these will be different for each face).

If you think the face showed no emotion you would choose 'neutral'.  
However, if you think there is an emotion in the face but you're not sure what it is, take a guess, but do not choose neutral.

(Press space to continue)

Then you will be asked how strong you think the emotion was.

Please choose a number from 0-7 to show this.

On this scale, 0 = no emotion, and 7 = the strongest the emotion could possibly be.

You can choose any number from 0-7.

(Please press space to continue)

Please look at the screen at all times during this task.

It is also very important that you sit as still as possible throughout this task.

(Please press space to continue)

Here are some examples of the types of faces you will be viewing.

Please practise rating these examples as requested on the screen.

(Press space to begin the practice items)

You have now completed the practice items.

Remember to look at the screen at all times during this task.

Please also stay as still as you can.

Feel free to ask any questions now.

(Please press space when you are ready to begin)

Thank you for completing this part of the study.

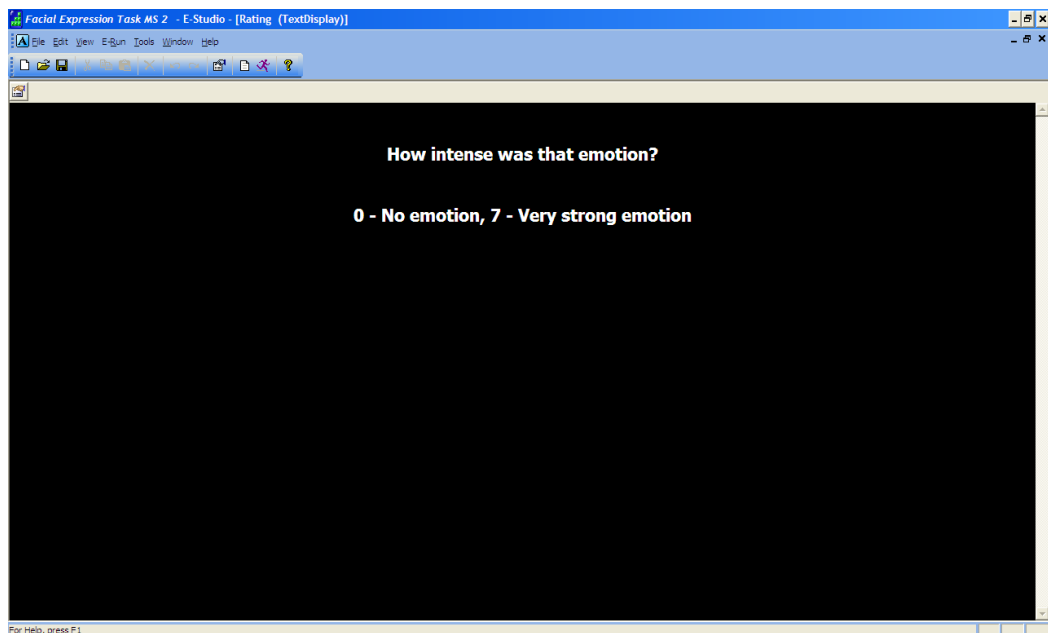
## **Appendix I6. Rating screens (facial expression processing task)**

### Emotion-labelling



Descriptors 1-5 = happiness, sadness, fear, anger, disgust, neutral (pseudorandomised on each trial)

### Intensity rating



## **Appendix 17. IAPS stimuli used in the affective picture viewing task**

### **Example stimuli**

Positive valence/low arousal



Positive valence/high arousal



Negative valence/low arousal



Negative valence/high arousal



Neutral



Practice stimuli: 1721, 2715, 7052










Experimental stimuli: 1300, 1441, 1610, 2397, 2399, 2490, 2540, 2590, 2811, 2840, 2880, 3030, 4608, 4660, 5001, 5551, 5621, 5623, 5760, 5833, 6830, 7006, 7041, 7175, 7230, 9001, 9090, 9300, 9331, 9405

**Appendix I8. Digitised self-assessment manikin (SAM) screen-shots**  
**(affective picture viewing task)**

**Valence**

How did that picture make you feel?










Unpleasant / Unhappy Pleasant / Happy

|                                                                                   |                                                                                   |                                                                                   |                                                                                   |                                                                                   |                                                                                   |                                                                                     |                                                                                     |                                                                                     |
|-----------------------------------------------------------------------------------|-----------------------------------------------------------------------------------|-----------------------------------------------------------------------------------|-----------------------------------------------------------------------------------|-----------------------------------------------------------------------------------|-----------------------------------------------------------------------------------|-------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------|
|  |  |  |  |  |  |  |  |  |
| 1                                                                                 | 2                                                                                 | 3                                                                                 | 4                                                                                 | 5                                                                                 | 6                                                                                 | 7                                                                                   | 8                                                                                   | 9                                                                                   |

**Arousal**

How did that picture make you feel?

Relaxed / Calm Tense / Excited

|                                                                                     |                                                                                     |                                                                                     |                                                                                     |                                                                                     |                                                                                     |                                                                                       |                                                                                       |                                                                                       |
|-------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------|
|  |  |  |  |  |  |  |  |  |
| 1                                                                                   | 2                                                                                   | 3                                                                                   | 4                                                                                   | 5                                                                                   | 6                                                                                   | 7                                                                                     | 8                                                                                     | 9                                                                                     |

## **Appendix 19. Standardised instructions (affective picture viewing task)**

You will now see many different pictures on the screen, one by one.

You will be asked to decide how each picture made you feel.

Please press space to continue.

Before each picture you will see a cross in the centre of the screen for some time.

Next, the picture will appear for a few seconds.

You will then be asked for two ratings about how the picture made you feel.

Please press space to continue.

One rating is about whether the picture made you experience pleasant or unpleasant feelings.

If you felt completely happy (pleasant, satisfied, contented, hopeful), press 9.

If you felt completely unhappy (annoyed, unsatisfied, bored), press 1.

You can also describe feelings in between, by pressing any number from 1 to 9.

If you felt completely neutral, select 5.

Please press space to see an example of the scale.

The other rating is about whether you felt calm or excited when looking at the picture.

The scale goes from feeling completely excited (stimulated, jittery, wide awake, aroused) to feeling completely relaxed (sluggish, sleepy or calm).

If you felt completely *aroused* or *excited* press 9.

If you felt totally relaxed and calm press 1.

Again, you can describe feelings in-between by pressing any number from 1 to 9.

If you did not feel at all excited or at all calm you would select 5.

Please press space to see an example of this scale.

Some of the pictures may make you feel strong emotions; others may seem neutral.

Please rate your immediate personal experience, and no more.

You will not always be asked to make your ratings in the same order.

Please press space to continue.

Please look at the screen at all times during this task.

Here are some examples of the types of pictures you will be viewing and rating.

Please practise rating these examples as requested on the screen.

Please press space to begin the practice items.

You have now completed the practice items.

Please remember to look at the screen at all times during this task.

Please make your ratings as quickly and accurately as possible.

It is also very important that you stay as still as you possibly can during the task.

Feel free to ask any questions now.

Please press space to begin.

Thank you for completing this part of the study.

## **Appendix 20. Semi-structured interview schedule (qualitative study)**

Can you describe to me, in general terms, what sort of a person you are emotionally?

Prompts:

Do your emotions/feelings change much over time?

Do you ever try to change your feelings?

Would you say that you generally experience more positive or negative emotions, or roughly the same amount of each?

Do your emotions ever affect aspects of your life? (e.g. your relationships, work/college)

Do you find it easy to name or label what you are currently feeling?

How do you tend to respond when you are faced with an emotionally upsetting event or situation?

Prompts:

For example, if somebody does something to upset or anger you?

Do you always know straight away when something has upset you?

Do you experience any physical changes when you are upset emotionally?

Do you tend to express your feelings?

Do you find it easy to calm down, once you are upset?

How easy do you find it to understand other people's emotional responses to things (feelings)?

Prompts:

Can you usually tell if somebody is upset, happy, sad, angry, etc?

If someone is upset or angry, can you generally understand why they may feel that way?

Do you ever feel confused by other people's reactions to things?

Do you feel that your emotions/feelings were involved when your seizures/attacks started?

Prompts:

- If yes, can you give me any ideas about what sort of events or experiences were most important in this?

- If no, what are your views on the reasons for why you started to have your seizures?

In your view, are emotions/stress involved in the fact that you are having your seizures?

Prompts:

If so, can you describe how you think this may be happening?

Do you feel that you have any control over this?

If emotions and stress are not reasons why you have seizures, why do you think that you are having seizures?

During, just before, or straight after your seizures, can you tell me if you regularly experience any specific emotions (have any particular feelings)?

Prompts:

E.g. joy, anger, or fear

Do you experience a sudden shift in the strength of your feelings/emotions?

Do you think that Dissociative Seizures are related to emotions/stress in general?

Prompts:

E.g. in other people

Is there anything else that you would like to tell me about your emotions and feelings? Particularly in relation to your diagnosis and experience of DS.